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DIVERTICULOSIS AND ACUTE DIVERTICULITIS OF THE JEJUNUM*

Report of Two Cases

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OUR interest in diverticulosis and diverticulitis of the jejunum was stimulated recently by the unexpected finding at laparotomy of such a case. A review of the records of the Faulkner Hospital from 1937 to 1949 reveals only 1 similar case. Because of the extreme rarity of this condition, these cases are reported below.

INCIDENCE

Jejunal diverticulosis is an uncommon finding. Edwards[1] described 5 cases in 881 autopsies — an incidence of 0.57 per cent. Browne and McHardy, according to Jones,[2] reported 16 cases in nearly 8000 autopsies. Roentgenologic study of the gastrointestinal tract has been a less sensitive indicator of the condition than autopsy because Case[3] found 5 cases in 6847 gastrointestinal series (0.073 per cent) and Edwards[1] 4 cases in 4631 barium-enema studies (0.086 per cent). Small-bowel diverticula, excluding Meckel's diverticulum, occur most frequently in the duodenum and upper jejunum, being far more common in the duodenum. They are rare in the ileum. In 1943 Benson, Dixon and Waugh[4] reported 122 cases of small-bowel diverticulosis from the Mayo Clinic, exclusive of duodenal and Meckel's diverticula. The jejunum was affected in 100 of these, and in 17 cases pouches were limited to the ileum. In 5 patients diverticula occurred throughout the small intestine. Diverticula were multiple in approximately half the cases. They occurred elsewhere in the body in 49 of 85 cases autopsied. The sites included the colon, duodenum, esophagus and urinary bladder.

The incidence of diverticulosis of the jejunum is equally distributed between the sexes. The patients are usually of middle or advanced age. However, the process may begin in early life, and the possibility of a congenital origin has been suggested by the high incidence of associated congenital anomalies.[4] These abnormalities include Meckel's diverticulum, malrotation of the intestine, hemangioma of the liver, exstrophy of the urinary bladder and congenital pyloric stenosis.

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PATHOGENESIS

Diverticula may be classified as true (congenital) and false (acquired). Meckel's diverticulum, with which we are not concerned in the present paper, is a true or congenital diverticulum; its walls are composed of all the layers of the normal intestine. The diverticula that concern us here are of the false or acquired type, although there may be a congenital weakness predisposing to their development. The walls of these false or pseudodiverticula lack the circular and longitudinal muscular coats of the normal bowel. Numerous investigators, among them Edwards,[1] have concluded that the pseudodiverticulum is a pulsion phenomenon occurring on the mesenteric border of the intestine at the site of penetration of the mesenteric vessels. Supporting this theory is the fact that the smaller pouches do not occur on the line of mesenteric attachment but rather just off that line on either side at the points where the vessels penetrate the muscular coats of the bowel. The smallest diverticula may have thinned-out muscularis. The walls of the larger ones are composed only of mucosa, submucosa and muscularis mucosae invested in serosa. Edwards[1] has demonstrated this amply by pathological sections. Grossly, the larger pouches appear paper thin and translucent. Large vessels course over the fundal portions parallel to the bowel. The stoma between diverticulum and bowel lumen varies with the size of the lesion and is generally large enough to permit free drainage.

SYMPTOMATOLOGY

There are no signs or symptoms pathognomonic of small-bowel diverticulosis or of any of its complications. Kiefer[5] discusses the difficulties inherent in the diagnosis of lesions of the small intestine. Symptoms occur only in the presence of

hypermotility or of obstruction. Hypermotility gives rise to diarrhea, a sense of fullness, vague supraumbilical cramps and increased flatulence. If hypermotility is persistent there may be increased bulk of stools and occasionally nutritional deficiency may occur. Owing to the liquid content of the small bowel, obstruction must be relatively severe to cause symptoms. Diverticula that empty themselves freely are usually silent. Mahorner and Kisner[6] state that duodenal diverticula give rise to symptoms more frequently than jejunal diverticula because of the fixed position with impaired emptying of the former. Crampy pain, intestinal noise and flatulence are the commonest symptoms recorded by numerous authors. Symptoms usually arise in the post-prandial period and subside spontaneously. Nausea may occur. Attacks may occur on successive days and may then disappear for long periods. In severe cases the symptoms may be incapacitating. Many such patients are doubtless labeled neurotic after studies of the gall bladder and upper gastrointestinal tract have ruled out the more common upper abdominal lesions. Small-bowel diverticula may be easily missed roentgenographically unless special care is taken in the study of the intestine.

Complications

Complications of jejunal diverticulosis include intestinal obstruction, hemorrhage, diverticulitis and rupture. Complications may be interrelated; for example, the adhesions of an old inflammatory process invite intestinal obstruction. Emphasis has been placed on the appearance of chronic intermittent obstruction without apparent cause, which occurs in cases of diverticulosis.[4]

Diverticulitis of the jejunum is less common than obstruction due to diverticulosis. Inflammatory changes range in degree from mild catarrhal states to gangrene, perforation and peritonitis. Generally, the inflamed pouch gives no clue to the cause of the process. The affected sacs usually drain freely, and contained fluid and gas are not under tension. It is not known whether intramural vascular thrombosis initiates the event. Free perforation is, of course, a catastrophe.

Diverticulitis may simulate a number of intraperitoneal inflammatory conditions. Judging from the paucity of reports of diverticulitis severe enough to warrant abdominal exploration, the condition must usually be mild. Successful preoperative diagnosis is reported by Shutkin[7] in a single case. Operation or autopsy alone provides the diagnosis as a rule. Abdominal spasm and collapsibility of diverticula usually prevent palpation of a mass. Other signs and symptoms are those common to all types of intraperitoneal inflammatory processes and are in no way diagnostic.

Treatment

Medical management of known cases of diverticulosis of the small bowel consists of a low-residue diet, mild laxation and post-prandial postural drainage. The effectiveness of any measures is uncertain. Complications already mentioned may necessitate surgical intervention. Surgical treatment has consisted of excision of solitary diverticula, enteroenterostomy and bowel resection. The first method is obviously not feasible in multiple diverticulosis. Bowel resection is poorly tolerated in depleted patients. Enteroenterostomy has been suggested by several writers, who believe bowel resection may be performed more advantageously at a later time. However, experience with the surgical treatment of small-bowel diverticula is so meager that no general rules can be stated.

The cases reported below illustrate many of the features outlined in patients with jejunal diverticulosis.

Case Reports

Case 1. A 77-year-old woman was admitted to the surgical service of the Faulkner Hospital on May 11, 1949, complaining of abdominal pain. The first symptoms were those of bloating and "dyspepsia." The patient had had such attacks occasionally for over 20 years. Such episodes had not been accompanied by nausea or vomiting or change in bowel habit. They had been sufficiently distressing 3 years earlier, however, to occasion x-ray studies of the gastrointestinal tract. At that time a diagnosis of diverticulosis of the duodenum and sigmoid colon was made. There was a suspicion of small-bowel diverticula, although this was not seriously entertained as a diagnosis. Gall-bladder function was normal by the Graham test. The patient was placed on a low-residue diet and given antispasmodics on the assumption that she had functional indigestion. After this regime she had fewer recurrences of discomfort, the most recent having been 8 months previously. Four hours prior to admission her usual vague indisposition gave way to symptoms of acute abdominal distress. After the passage of a normal stool, nausea and vomiting began, and at the same time steady lower abdominal pain developed. This gradually increased in severity and was aggravated by bodily movement. There was no radiation of the pain.

Physical examination was not remarkable except for some evidence of dehydration and a Grade III aortic systolic murmur. The right side of the abdomen was more prominent than the left, but no palpable mass could be made out, and there was no visible peristalsis. There was tenderness in the right upper quadrant and across the lower abdomen, more on the right, with some spasm in this region. There was rebound tenderness throughout the lower abdomen. Pelvic examination revealed tenderness in both vaults and pain on motion of the uterus.

The temperature was 100.2°F., the pulse 80, and the respirations 20.

The blood pressure was 148/90.

The white-cell count was 15,600, with 95 per cent neutrophils. The urine gave a ++ test for albumin, a + test for sugar and a + test for acetone, and the sediment contained a few hyaline casts. A plain film of the abdomen showed no dilated bowel, no free air and no fluid levels. Fluid-replacement therapy was instituted. Three hours after admission the physical signs had become more localized in the right lower quadrant, and an exploratory laparotomy was carried out with a preoperative diagnosis of acute appendicitis.

The abdomen was opened through a pararectus incision in the right lower quadrant. A small amount of cloudy, straw-colored, odorless fluid was encountered. This was cultured and later proved to be sterile. Presenting in the wound was a loop of upper jejunum showing multiple diverticula, scattered along for a distance of about 60 cm. Their number

was estimated at 50, and they varied from 1 to 10 cm. in diameter. They arose from points adjacent to the mesentery, and the walls of the larger ones were translucent and paper thin; all emptied easily on pressure. Near the center of the involved area a 3-cm. diverticulum was acutely inflamed and fibrin coated, and the adjacent mesentery was thick, edematous and indurated. There was no evidence of perforation.

Because of the extensive inflammatory reaction in the mesentery, a resection was not considered wise in this elderly patient. A grossly normal appendix was removed, and the abdomen closed. She made an uneventful recovery and was discharged on the 14th day after operation. Barium x-ray studies before discharge showed multiple diverticula of the duodenum and jejunum (Fig. 1), with 24-hour retention of barium in several of them.

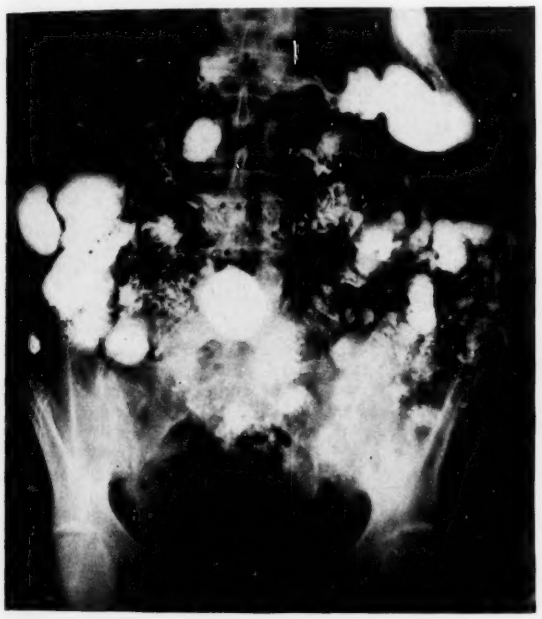

FIGURE 1. *Multiple Barium-Filled Diverticula of the Duodenum and Jejunum.*

On a low-residue diet and antispasmodics, the patient did fairly well for 2 months and then had another similar attack, which subsided in the hospital on conservative measures. Subsequently, however, her recurrent attacks of "bloating" were worse and were quite incapacitating. Four months after the first attack she was again admitted to the hospital, and a resection of the involved segment of jejunum was carried out. There was clear evidence of the previous acute inflammatory process. The resected specimen is shown in Figure 2. All the sizable diverticula of the small bowel that could be found on careful exploration were included in this 60-cm. segment starting just distal to the ligament of Treitz. Scattered in the lower jejunum were a number of 1-cm. diverticula. None were found in the ileum. She made an uneventful recovery and was discharged from the hospital in 2 weeks. She was symptom free in the hospital, but a prolonged follow-up period is needed to determine whether or not the source of the chronic symptoms has been removed.

CASE 2. A 36-year-old mother of 3 children was admitted to the Faulkner Hospital on December 4, 1937, with a history of vomiting and crampy generalized abdominal pains beginning after a large meal taken 18 hours previously. No previous similar episodes had occurred, and she had had no digestive complaints in the past. The pain passed in waves from the upper to the lower abdomen and was not relieved by vomiting. There had been no bowel movements or passage of flatus.

Physical examination on admission showed a well nourished woman in moderate distress. The abdomen was diffusely tender without localization. There was spasm, particularly in the left flank anteriorly. Peristalsis was normal. Pelvic and rectal examinations were normal.

The temperature was 100.4°F. The white-cell count was 25,000, with 95 per cent neutrophils, and the urine was normal. Exploratory laparotomy was carried out, a mass in the left upper quadrant being encountered, which proved to consist of omentum adherent to the jejunum and its mesentery. An acute inflammatory reaction with abundant fibrin was present. Upon detachment of adherent omentum, purulent fluid appeared from a 3-cm. sac at the juncture of mesentery and bowel wall. Gas could be expressed easily from the bowel into this sac. A 9-cm. segment of involved jejunum was resected, and an end-to-end anastomosis was performed. Gross and microscopical study of the resected segment established the diagnosis of an acutely inflamed diverticulum of the jejunum with perforation intc the mesentery.

Postoperative signs of high intestinal obstruction appeared, and on the 5th postoperative day laparotomy and jejunostomy were performed to circumvent the nonfunctioning end-to-end anastomosis. A feeding jejunostomy completed the operation, and recovery thereafter was uneventful. The patient was discharged on the 31st hospital day.

FIGURE 2. *Resected Portion of the Upper Jejunum. The multiple diverticula of varying sizes are clearly seen.*

She succumbed to a rapidly growing spongioblastoma multiforme in the following year. At post-mortem examination five diverticula measuring 2.5 cm. in diameter were found in the jejunum. They were on the mesenteric aspect of the gut. No other intestinal diverticula were found.

SUMMARY

Two cases of acute diverticulitis of the jejunum are reported, and some aspects of this rare condition are discussed.

Diverticulitis of the jejunum should be considered as a diagnostic possibility in acute abdominal conditions, particularly in elderly people.

Diverticulosis of the jejunum is an occasional cause of digestive symptoms.

References

1. Edwards, H. C. Diverticulosis of small intestine. *Ann. Surg.* **103**: 230-254, 1936.
2. Jones, C. M. Gastroenterology: review of literature from July 1939 to July 1940. *Arch. Int. Med.* **66**:893-1004, 1940.
3. Case, J. T. Diverticula of small intestine, other than Meckel's diverticulum. *J. A. M. A.* **75**:1463-1470, 1920.
4. Benson, R. E., Dixon, C. F., and Waugh, J. M. Non-meckelian diverticula of jejunum and ileum. *Ann. Surg.* **118**:377-393, 1943.
5. Kiefer, E. D. Clinical aspects of chronic disease of small intestine. *J. A. M. A.* **113**:1546-1552, 1939.
6. Mahorner, H., and Kisner, W. Diverticula of duodenum and jejunum. *Surg., Gynec. & Obst.* **85**:607-622, 1947.
7. Shutkin, M. W. Diverticulitis of jejunum with perforation. *Gastroenterology* **5**:102-105, 1945.

AMEBIC PERICARDITIS*

Review of the Literature and Report of a Case

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AMEBIC pericarditis is a rare complication of infection with *Endamoeba histolytica*. A review of the literature from 1885 to the present revealed 44 proved cases of suppurative amebic pericarditis. Pericarditis was reported to have developed in 14 additional patients with known amebiasis. In these, however, the diagnosis was established either by the finding of serous fluid in the pericardial sac at post-mortem examination or by the appearance, during life, of a pericardial friction rub that disappeared after treatment with emetine hydrochloride. In all cases the pericardial involvement was secondary to an amebic abscess adjacent to the pericardial sac in either liver or lung.

Ochsner and DeBakey[1] reported that liver abscesses developed in 4.8 per cent of over 9000 persons infected with *E. histolytica*. Rupture of liver abscesses occurs most commonly into the pleura, lung or peritoneum and more rarely into the pericardial sac or an abdominal viscus. Direct rupture of an amebic abscess through the pericardium occurred in 2.8 per cent of 689 cases of perforated liver abscesses reported by Vergoz and Hermenjat-Gerin.[2] The rarity of pericardial involvement in patients with clinical amebic infection is therefore apparent.

The diagnosis of amebic pericarditis was established during life in only 2 of the 44 proved cases. In neither case was the pericardial sac drained directly, although 1 patient recovered after indirect drainage of this cavity through the liver abscess. The purpose of this report is to present a case of suppurative pericarditis secondary to rupture of a liver abscess in which the amebic etiology was established after surgical drainage of the pericardial sac.

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Case Report

A 24-year-old Hindu seaman entered the hospital on August 19, 1948, complaining of retroxiphoid and epigastric pain.

The patient had been well until 1 month prior to admission, when he noted the onset of moderate anorexia. Two weeks later, he felt a sharp pain in the left shoulder anteriorly, which persisted as an ache, was aggravated on deep inspiration and was not affected by movement of the joint. Twelve days prior to admission chills developed, accompanied by a feeling of feverishness and a dry mouth. One week before admission, a dull, persistent retroxiphoid and epigastric pain appeared and soon spread to the left anterior costal margin and was more severe on deep inspiration. Drenching night sweats and an infrequent dry cough developed during the 5 days prior to admission. On the day of entry, while walking on the street, he suddenly became weak, collapsed without loss of consciousness and was brought to the hospital.

The patient stated that he had been born in Portuguese Goa, India, had served in the British Navy for 5 years during the war, and for the past 3 years had lived in the United States continuously. He gave no history of diarrhea or other gastrointestinal symptoms occurring at any time during his life. The remainder of the system review was not significant.

The family history was noncontributory. Two sisters were living and well. The father and mother had died of a fever of unknown type 11 years prior to the patient's illness.

Physical examination revealed a well developed, poorly nourished man who was acutely and chronically ill and complaining bitterly of pain in the left shoulder anteriorly. The pharyngeal mucosa was slightly reddened. The skin was hot and moist. There was moderate rigidity and tenderness in the upper abdomen, which was most pronounced in the mid-epigastrium. Neither the liver nor the spleen could be percussed or palpated below the costal margins. The peristaltic sounds were normal, and there was neither abdominal distention nor costovertebral-angle tenderness. The heart and lungs were normal, and the remainder of the physical examination was not significant.

The temperature was 99.2°F., the pulse 90, and the respirations 20; the blood pressure was 110/70.

Examination of the blood showed a hemoglobin of 9.0 gm per 100 cc. and a hematocrit of 36 per cent. The white-cell count was 10,000, with 50 per cent neutrophils, 38 per cent lymphocytes, 4 per cent atypical lymphocytes and 8 per cent monocytes. Urinalyses demonstrated a normal concentration and no sugar or albumin, but occasional white cells and granular casts were present in the centrifuged sediment. The blood Hinton test was negative. The blood nonprotein nitrogen level was 32 mg. per 100 cc. A stool was brown, formed and guaiac negative and contained no ova or parasites. A blood culture taken on admission showed no growth. A lumbar puncture performed on the day following admission revealed a normal cerebrospinal fluid.

Initial roentgenograms of the left shoulder were interpreted as showing no abnormality. An x-ray film of the chest showed questionable prominence of the left ventricle.

Chills occurred daily during the first 2 weeks, and the temperature was irregularly elevated, ranging from 99.0 to 103.0°F. The patient took a soft diet poorly and was given fluids by mouth, barbiturates for sleep and codeine and aspirin to control the persistent pain in the left shoulder and epigastrium. No specific diagnosis was established, but miliary tuberculosis was considered most tenable.

Further laboratory determinations showed a total white-cell count varying from 12,550 to 16,350, and multiple blood smears revealed 61 to 66 per cent neutrophils, 30 to 35 per cent lymphocytes, 2 per cent monocytes and 1 to 6 per cent eosinophils. Examination of the stained blood film demonstrated no malarial parasites. The urine remained normal except for the presence of occasional white blood cells in the sediment. Urine cultures were sterile, as were repeated blood cultures. Agglutination tests of the patient's serum for *Salmonella typhi*, *S. paratyphi* (B), *S. cholerasuis*, polyvalent dysentery organisms and *Brucella abortus* were all negative. The Weil–Felix reaction was negative. Heterophil-antibody agglutination was within normal limits.

On the 15th hospital day, a dull, constant pain occurred in the anterior portion of the right shoulder. This pain was more intense on inspiration, and its character was described by the patient as similar to the persisting pain in the left shoulder. On the following day a harsh pericardial friction rub could be heard over the entire precordium. Intramuscular injection of penicillin was then begun in doses of 50,000 units every 3 hours.

A roentgenogram of the chest at this time showed no change from the appearance on admission. An electrocardiogram, recorded on the day after the friction rub appeared, showed only minimal T-wave changes. The temperature continued to rise daily to 101°F., and the pulse rate varied from 90 to 110 beats per minute. A roentgenogram of the chest on the 23rd hospital day showed an extensively enlarged cardiac shadow. An electrocardiogram was consistent with the diagnosis of pericarditis (Fig. 1). Distended neck veins, a paradoxical pulse and a diminished pulse pressure were also observed.

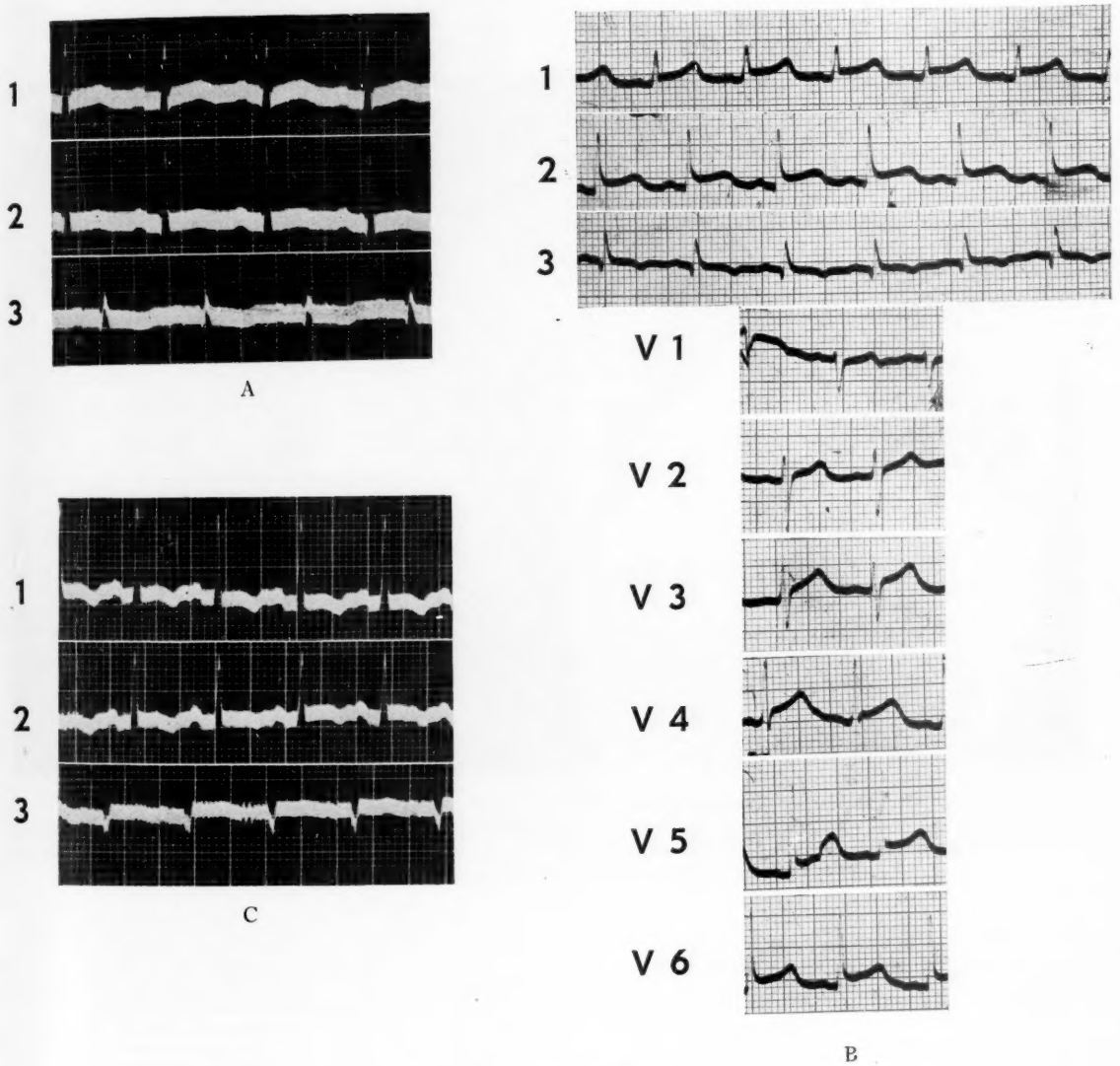

FIGURE 1. *Electrocardiograms.*

A = tracing taken the day after the appearance of a friction rub, showing a borderline T wave in Lead 2. B = tracing taken on the twenty-third hospital day, showing a pattern consistent with the diagnosis of acute pericarditis. C = tracing taken on the thirty-second hospital day, showing evolution of the pericarditic pattern.

Aspiration of the pericardial sac through a needle inserted in the left costoxiphoid angle yielded 100 cc. of pale-orange, purulent fluid, which was creamy in consistence. Examination of the stained sediment from this fluid revealed 150 white blood cells and 50 red blood cells per high-power microscopic field. No organisms were found in smears of this pus, stained by both the Gram and Ziehl–Neelsen technics. Bacteriologic cultures of the fluid were sterile.

Clinical improvement was observed after the pericardial aspiration. This was demonstrated by a fall in temperature, a slower pulse rate and disappearance of the venous distention. Improvement continued for 2 days, when signs of severe cardiac tamponade returned. Aspiration of the pericardial sac was repeated daily for the following 4 days. Fluid similar to that previously withdrawn was obtained in amounts varying from 300 to 450 cc. No organisms were demonstrated by either direct smear or culture of the fluid.

Penicillin, in a dose of 100,000 units, was instilled in the pericardial sac after each of these aspirations. Parenteral administration of penicillin was increased to 500,000 units every 3 hours.

The patient continued to complain of severe epigastric and bilateral shoulder pain, which improved temporarily after each pericardicentesis. He ate poorly, lost weight and looked increasingly ill. On the 31st hospital day, 8 days after the first aspiration, the pericardial fluid was found to be red brown and somewhat thicker. Three hundred cubic centimeters of fluid was removed, 60 cc. of air was instilled, and roentgenograms were taken with the patient in various positions to map the pericardium. Pneumopericardiograms demonstrated a thickened pericardium and showed no loculations within the sac (Fig. 2).

Surgical drainage of the pericardium was decided upon because the increasing viscosity of the pericardial fluid prevented adequate relief of the cardiac tamponade by aspiration. Furthermore, a sinus tract developed in the costoxiphoid angle, the site of multiple pericardicenteses. On the 32nd hospital day, the pericardium was opened by resection of the sternal cartilages of the fourth and fifth ribs on the left side. Local anesthesia was used. The pericardium was marsupialized to the skin of the anterior chest wall. Approximately 1 liter of thick fibrinous creamy material was removed. Both the pericardium and epicardium were thickened, and each was covered by a shaggy, greenish-white deposit of fibrin and pus. The blood pressure, which was 80/70 at the beginning of the operation, rose sharply to 120/80 on release of the tamponade. The pathological report of a biopsy of the pericardium taken at operation disclosed "acute and chronic inflammation in granulation tissue and organizing hemorrhage."

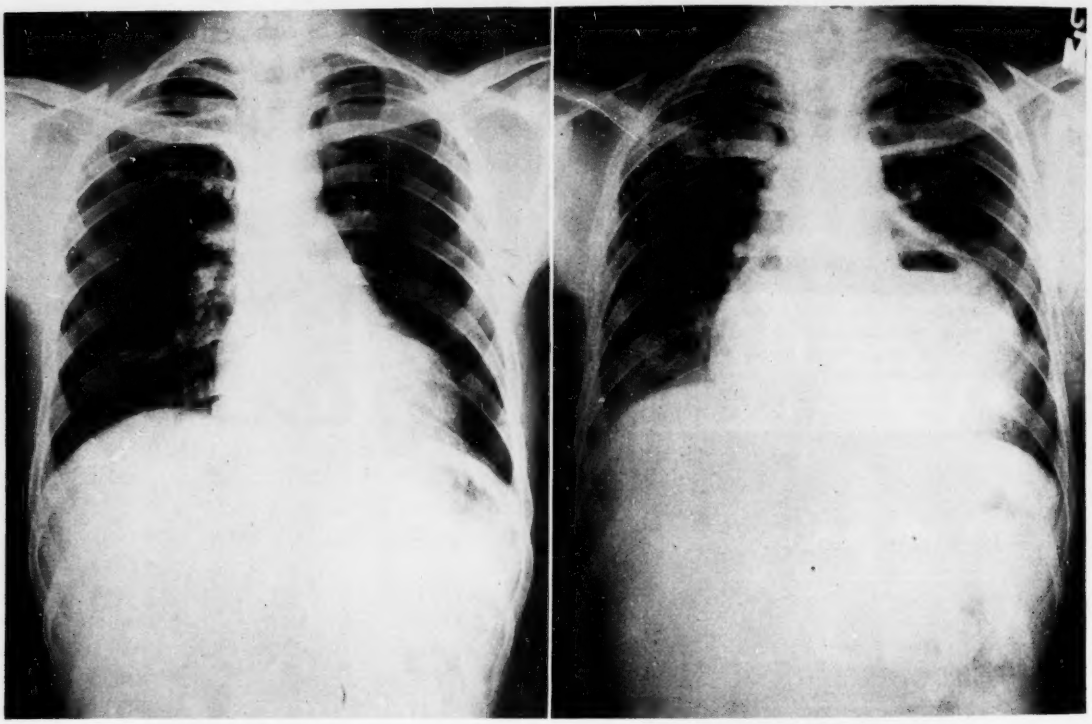

FIGURE 2. *Roentgenograms of the Chest in a Case of Amebic Pericarditis.*
The film on the left, taken on admission, shows only questionable enlargement of the left ventricle. The film on the right, taken after pericardial aspiration and injection of air on the thirty-first hospital day, demonstrates an enlarged pericardial shadow, thickened pericardial wall and fluid level in the pericardial sac.

The patient improved subjectively after the operation. The improvement in the blood pressure was maintained. However, temperatures varied from 99 to 101°F. by rectum, and the pulse rate remained at approximately 120 per minute. The patient ate poorly. A moderate bilateral pleural effusion, which was greater in the right hemithorax developed, and thoracentesis performed on this side yielded 350 cc. of clear, serous, sterile fluid. The pericardial cavity was irrigated every 4 hours with half-strength Dakin's solution, and the fibrinous cardiopericardial adhesions were removed digitally once daily. Several blood transfusions were given, and the hematocrit was 42 per cent and the hemoglobin 12 gm. per 100 cc. 1 week after the operation.

On the 8th postoperative day, during the digital exploration of the pericardial cavity, a soft area was felt on the floor of the pericardium posteriorly. The examiner's finger fell through this into a large cavity from which welled a considerable quantity of yellow-green, foul-smelling fibrinopurulent material. At that time the diagnosis of liver abscess was established.

Examination of fresh material from the cavity revealed myriads of organisms identified as *E. histolytica* (Fig. 3).

Treatment was then begun with emetine hydrochloride in a daily intramuscular dose of 0.06 gm. (1 gr.) The need for adequate dependent drainage of the liver abscess was realized as the patient's condition grew worse. Therefore, on the 10th postoperative day, the liver abscess was drained through a left subcostal approach, local infiltration anesthesia being used.

FIGURE 3. *Photomicrograph of Fluid from the Pericardial Sac. (Paraffin-Block Section, Iron-Hematoxylin Stain, x1600.) Note the large trophozoite of* E. histolytica, *with an eccentric nucleus, a nucleolus and parachromatin granules just within the nuclear membrane. The cytoplasm contains many ingested red blood cells. The trophozoite is surrounded by many chronic inflammatory cells.*

On the following morning (the 45th hospital day), the patient's condition appeared unchanged. However, just before his wound was dressed, he suddenly died. No immediate cause of death was apparent.

Permission for autopsy was obtained with difficulty. It was learned that the patient had deserted a cargo ship and, contrary to his assertion, had been living in the United States only 6 weeks prior to admission to the hospital.

Post-mortem examination, performed 6 weeks after death, revealed two small liver abscesses. One, measuring 5 cm. in diameter, was situated in the left lobe of the liver directly beneath and communicating with the pericardial sac through an aperture approximately 1.5 cm. in diameter. A second abscess, measuring 4 cm. in diameter, was situated in the lower portion of the right liver lobe near the anterior surface (Fig. 4). The opened pericardium was thickened, shaggy and adherent to the anterior chest wall. The pericardial sac contained about 20 cc. of thick, gray fluid. There were no adhesions to the heart. The epicardium was thickened and fibrotic, but the heart was otherwise normal. Approximately 300 cc. of serous fluid was present in each pleural cavity. Both lungs were fixed to the mediastinum and to the surface of the diaphragm by dense fibrous adhesions. Neither the lungs nor the mediastinum was otherwise remarkable. Examination of the entire gastrointestinal tract revealed no evidence of disease.

Extreme post-mortem degeneration was evident in all the tissues on microscopic examination. The pericardium was thickened, edematous and infiltrated with mononuclear cells and numerous degenerated trophozoites of *E. histolytica*. The epicardium was thickened by a superficial fibrin layer, which contained degenerated trophozoites and by a subjacent organized layer infiltrated with mononuclear cells. A slight increase of loose connective tissue and a scattered infiltration with lymphocytes were present in the myocardium. The pleura was slightly edematous and fibrotic. The alveolar spaces contained pigment-bearing macrophages, a few red blood cells and round cells. No trophozoites were found in either the lungs or the pleura. The walls of the liver abscesses were necrotic and contained a few inflammatory cells and many degenerated trophozoites. No significant pathological changes were evident in the microscopic sections of the remaining organs.

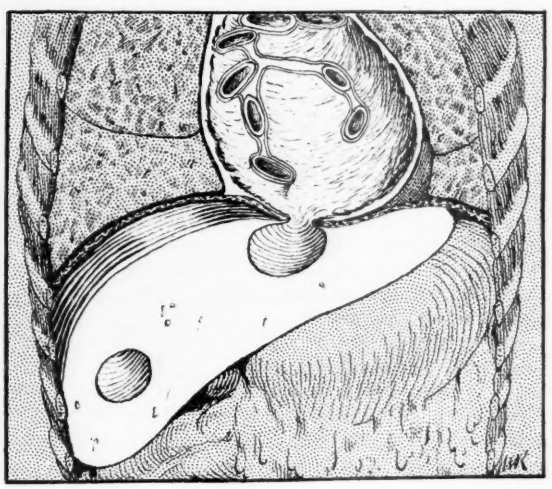

FIGURE 4. *Artist's Sketch of Gross Findings at Autopsy. Note the communication between the pericardial sac and the abscess in the left lobe of the liver.*

DISCUSSION

Table 1 summarizes the available information on 58 reported cases of pericarditis associated with amebic infection.* Only 1 of the 44 patients with proved cases of suppurative pericarditis survived.[9] In that case, the diagnosis of pericarditis was established by pericardial aspiration. Recovery followed treatment with emetine hydrochloride and surgical drainage of an abscess in the left lobe of the liver underlying the pericardium. Singh[14] reported the only other case of amebic pericardial suppuration diagnosed during life. This patient did not survive despite treatment with emetine hydrochloride and needle aspiration of the pericardial sac.

The primary complaints of epigastric, infracostal and left-shoulder pain form some substantial basis

*The report of Staffieri et al.[16] was not available for review.

for a possible diagnosis of liver abscess. The persistent evidence of infection, the pain from diaphragmatic irritation, the absence of pulmonary findings and the negative bacteriologic studies suggest liver involvement. Because of the patient's race and foreign origin, enteric pathogens were sought, and he was carefully questioned about antecedent dysentery. However, a negative history is not always significant since half to two thirds of all patients with hepatic amebiasis give no history of antecedent diarrhea.[17] Furthermore, liver involvement has occurred as late as twenty to forty-three years after the primary symptoms of diarrhea due to amebas.[1] It has been shown that only 11 to 36 per cent of patients with amebic hepatic abscesses pass amebas in their stools.[1] Even a series of negative stool examinations does not exclude the diagnosis of hepatic amebiasis. Failure to find amebas in aspirated fluids does not exclude the diagnosis either, since the organisms are present in the fluids of only 16 per cent of liver abscesses caused by *E. histolytica*.[1]

The complement-fixation test is valuable, and should have been performed in the case reported above. This test may be negative in the carrier or asymptomatic patient, but it is positive in 90 per cent of patients with hepatic or pulmonary amebiasis.[17]

In 5000 cases of amebic liver abscesses collected from the literature, treatment by aspiration resulted in a mortality of 5.6 per cent, whereas treatment by open drainage resulted in a mortality of 43.1 per cent. Open drainage is recommended only for advanced liver abscesses that are secondarily infected at the time of diagnosis.[1] The importance of early diagnosis is apparent. Adequate treatment should result in cure of most of these patients before perforation of the liver abscess occurs.

TABLE 1. *Data in 58 Cases of Pericarditis Associated with Amebic Infection.*

AUTHOR	YEAR	NO. OF CASES	CASES OF PERICARDITIS		DIAGNOSIS DURING LIFE	RESULT	
			SUPPURATIVE	NON-SUPPURATIVE		RECOVERY	DEATH
Howard and Hoover[3]	1897	1	1		No	0	1
Craig[4]	1924	12	2	10	No	0	12
Justi[5]	1923	19	19		*	*	*
Surbek[6]	1928	1	1		No	0	1
Laigret[7]	1928	1		1	Yes	1	0
Kataropulo[8]	1932	1	1		No	0	1
Vergoz and Hermenjat-Gerin[2]	1932	13	13		*	0	13
Huard and Meyer-May[9]	1936	3	3		Yes (1 case)	1	2
Purcell[10]	1938	1		1	Yes	1	0
Ochsner and DeBakey[1]	1943	1	1		No	0	1
Herrlich[11]	1943	1	1		No	0	1
Kern[12]	1945	1	1		No	0	1
Laha[13]	1946	1		1	Yes	1	0
Singh[14]	1946	1	1		Yes	0	1
Edwards[15]	1947	1		1	Yes	1	0

*Information not available.

This paper reports a fatal case of suppurative amebic pericarditis that was diagnosed during life after pericardiostomy.

SUMMARY

A review of the literature has revealed 44 cases of suppurative pericarditis caused by *Endamoeba histolytica*. Pericarditis is reported to have developed in 14 additional patients with known amebiasis.

REFERENCES

1. Ochsner, A., and DeBakey, M. Amebic hepatitis and hepatic abscess: analysis of 181 cases and review of literature. *Surgery* **13**: 460-493 and 612-649, 1943.
2. Vergoz and Hermenjat-Gerin. De la rupture des abcès amibiens du foie dans les cavités séreuses (plèvre, péritoine, péricarde). *Rev. de chir.* **51**:680-734, 1932.
3. Howard, W. T., Jr., and Hoover, C. F. Tropical abscess of liver, with consideration of its pathology and clinical history. *Am. J. M. Sc.* **114**:150-166, 1897.
4. Craig, C. F. Complications of amoebic and specific dysentery as observed at autopsy: analysis of one hundred and twenty cases. *Am. J. M. Sc.* **128**:145-156, 1904.
5. Justi, K. Metastische Amöbenerkrankungen. In *Handbuch der Tropenkrankheiten*. Edited by C. Mense. Second edition. 4 vol. Leipzig: J. A. Barth. 1923. Vol. 1. Pp. 58-137.
6. Surbek, K. E. Ein Fall von Amöbenpericarditis. *Arch. f. Schiffs-u. Tropen-Hyg.* **34**:456, 1930.
7. Laigret, J. Péricardite au cours d'une dysenterie amibienne. *Bull. Soc. path. exot.* **21**:753-755, 1928.
8. Kataropulo, A. Amöbenperikarditis. *Arch. f. Schiffs-u. Tropen-Hyg.* **36**:544-548, 1932.
9. Huard, P., and Meyer-May, J. *Les abcès du foie*. 390 pp. Paris: Masson et Cie, 1936.
10. Purcell, F. M. Case of amoebic hepatic abscess and associated pericarditis. *Tr. Roy. Soc. Trop. Med. & Hyg.* **31**:689, 1938.
11. Herrlich, A. Ein Fall von linksseitigen Leberabscess mit Durchbruch ins Pericard. *Wien. klin. Wchnschr.* **56**:431-434, 1943.
12. Kern, F., Jr. Amebic pericarditis. *Arch. Int. Med.* **76**:88-92, 1945.
13. Laha, P. N. Amoebic hepatitis and associated pericarditis. *Indian M. Gazette* **81**:528, 1946.
14. Singh, G. Amoebic abscess of liver bursting into pericardial cavity. *Indian M. Gazette* **81**:299-301, 1946.
15. Edwards, M. L. Amoebic pericarditis. *M. J. Australia* **1**:177, 1947.
16. Staffieri, D., Gonzalez Sabathie, L., and Kruse, H. A. Pericarditis y absceso amebiano del hígado. *Rev. méd. de Rosario* **34**:118-122, 1944.
17. Elsom, K. A. Amebiasis, with especial reference to its complications. *Veterans' Administration Technical Bulletin* **10**-27, April 12, 1947.

PROBLEMS AND PRACTICES IN A COMMUNITY HOSPITAL*

I. Treatment of Venous Thrombosis

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GARDNER, MASSACHUSETTS

AT THE Henry Heywood Memorial, a 120-bed general hospital, during 1947-1948 there were 8129 admissions. Among these were 60 cases of diagnosed venous thrombosis, or approximately 1 case in each 135 admissions. Since this type of hospital cares for about half the general patients in this state,[1] the problem of treating venous thrombosis must be faced here as surely as in the large teaching centers.

At present, the proper method of treating venous thrombosis is controversial. The literature on the subject in the past five years is prima-facie evidence of this statement. The physician who is faced with the immediate problem at the bedside all too frequently believes that he must choose between methods, each of which has its unyielding advocates.

How then are these patients with venous thrombosis best treated? In attempting to answer this question, we do not pretend to offer anything original. We are merely presenting a consolidation of ideas derived from the literature and from our own experience and that of others. From this we have derived an orderly plan of attack, adaptable to our local situation.

Before continuing, we wish to emphasize the definition of two terms needed to provide a common ground for discussion. In our management of venous thrombosis, we consider it important to distinguish *phlebothrombosis* from *thrombophlebitis*. Some have erroneously used the term thrombophlebitis to indicate both conditions. We find it difficult to apply one term to two manifestations of venous thrombosis that differ so completely clinically and pathologically. It has been pointed out[2-4] that thrombophlebitis is an acute inflammatory process associated with a fixed clot and easy to diagnose by the presence of heat, swelling and tenderness. The danger of fatal embolism is slight. Phlebothrombosis, on the other hand, is a bland, noninflammatory process, undiagnosed except by the most diligent, repeated examinations and often suspected only after pulmonary infarction has occurred. The diagnosis is suggested by slight, unexplained fever, slight rise in pulse and respiration, minimal swelling of the ankles and calf tenderness, often not sufficient to elicit a voluntary complaint on the part of the patient. Here the clot lies silent and may strike with sudden unexpectancy to produce a vascular catastrophe. It is common knowledge that both conditions may exist coincidentally in the same patient. A strict acceptance of these terms in the true sense of their meanings has been helpful to us in visualizing the underlying lesion of each case problem.

The diagnosis of venous thrombosis is not within the province of this paper, but we believe it important to mention certain observations that have had a bearing on our treatment. As is often the case, the early diagnosis of a disease is made by recognition of the possibility of its presence. We have found that delay in diagnosis has resulted in some cases from failure to look for the disease in the veins rather than from actual failure of diagnosis when careful observations have been performed. In some cases in which pulmonary infarction was the first recognized sign, we have found on close questioning of the patient that he did have either slight cramps in the legs or a heaviness in one of the extremities, not sufficiently acute to make him regard it as important to mention. Because of this, we feel duty bound in every suspected case to examine the legs closely every day.

When the practice of early ambulation was first started in this hospital late in 1945, it was believed that it might reduce the incidence of venous thrombosis. Certainly on a theoretical basis this would be expected, and many authors[5-9] regard early ambulation as a desirable prophylactic measure, some[10-12] even considering it of primary importance. Other investigators[13-16] have been unable to show any decreased incidence of venous thrombosis in patients who are permitted to rise early. There has been considerable confusion and lack of agreement about what constitutes early ambulation. In our hospital the practice consists in actually walking the patient, or having him mark time at the bedside, starting in the first twenty-four to thirty-six hours. He does not sit up until he is ambulatory without assistance. Although early rising has proved itself an invaluable adjunct to the postoperative care of the patient, we are unable to find any statistical comfort from our experience with early ambulation in venous thrombosis.

It is our further opinion that anticoagulant therapy and venous interruption are not competitive methods of treatment and that it is not mandatory to choose one method and use it to the exclusion of the other, but rather that they should be used singly or in combination as indicated.

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Finally, it must be emphasized that venous thrombosis is often erroneously considered only a postoperative complication. It is, however, more common in medical patients; thus, Carlotti and his associates[17] report that over 50 per cent of pulmonary emboli occur in medical patients.

Against the background of the foregoing concepts we have arrived at the following method of treating venous thrombosis (Table 1) in our small community hospital.

TABLE 1. *Indications for Prophylactic and Therapeutic Procedures in the Management of Venous Thrombosis.*

CONDITION	ANTICOAGULANT TREATMENT	VENOUS INTERRUPTION
Prophylactic indications*:		
Medical:		
Myocardial infarction	Indicated	Not used
Prolonged bed rest of patients with previous history of venous thrombosis	Indicated	Not used
Surgical:		
Elderly patients with:		
Major amputations or severe trauma to bones or soft tissues of the lower extremities	Not used	Indicated
Extensive gastrointestinal surgery	Not used	Indicated
Stage operations	Not used	Indicated
Therapeutic indications*:		
Medical:		
Prolonged enforced bed rest in any patient who develops signs of venous thrombosis or its complications (congestive heart failure, pneumonia, prolonged infections)	Indicated	Not used
Obstetric:		
Venous thrombosis or its complications in:		
Ante-partum cases	Contraindicated	Indicated
Post-partum cases	Indicated	
Surgical:		
Phlebothrombosis with mild to moderate or without pulmonary involvement	Indicated	Not used
Phlebothrombosis with massive pulmonary infarction	Indicated†	Indicated†
Thrombophlebitis (to guard against phlebothrombosis in opposite leg)	Indicated	Not used
Cases with venous thrombosis in which there is:		
Impaired liver or kidney function	Contraindicated	Indicated
Blood dyscrasias	Contraindicated	Indicated
Surgery in which hemorrhage or oozing may be postoperative factor	Contraindicated	Indicated
Active bleeding from any source (peptic ulcer, uterine tumor)	Contraindicated	Indicated
Failure of response to anticoagulants		Indicated
Neurologic disease or injury to central nervous system	Contraindicated	Indicated
Prolonged use because of expense and impracticability	Contraindicated	Indicated
Prolonged use of acetylsalicylic acid, as in arthritis with potential hypoprothrombinemia (not common)	Contraindicated	Indicated
Patients with pulmonary infarction:		
When heparin is not immediately available	Contraindicated	Indicated
Patients with repeated emboli in spite of anticoagulants		Indicated

*If laboratory facilities are inadequate, all patients with venous thrombosis or its complications should have venous interruption.
†Veins interrupted as emergency measure with simultaneous administration of anticoagulants.

ANTICOAGULANT TREATMENT

Anticoagulants have proved to be an effective tool in the treatment of venous thrombosis. The nature, source, mode of action and rationale for the use of the most common anticoagulants, heparin and Dicumarol, have been extensively studied and reported,[18, 19] and any review here would be repetitious. It cannot be overemphasized, however, that the use of Dicumarol requires adequate laboratory control through performance of daily prothrombin determinations. This implies the service of technicians who have developed more than ordinary skill in doing the test, through repeated execution, and who are thereby well acquainted with its many pitfalls. Since the actual prothrombin time in seconds varies with each test, the result should be expressed in percentage for uniformity. Furthermore, the clinician administering the anticoagulant should have a thorough understanding of the test in use since the desired therapeutic levels differ with almost each type of test. One of us (T. J. D.), at another hospital, using a test employing thromboplastin, sought to achieve a 20 to 40 per cent prothrombin activity for therapeutic effect. Here, however, with the use of the viper-venom technic,[20, 21] a therapeutic level is reached when the prothrombin percentage is between 45 and 60. By this method the actual prothrombin time is 25 seconds, and this is considered to be 100 per cent. Therefore, on a direct-proportion ratio, a prothrombin time of 50 seconds would be reported as 50 per cent. This corresponds to about 20 per cent prothrombin activity as determined by actual serial dilution of a 100 per cent sample of plasma.[22] By whatever method used it is desirable to maintain the actual prothrombin time, in seconds, at twice the control (50 seconds or 50 per cent by our tests). On this basis, then, we consider the therapeutic range as being 45 to 60 per cent.

When it has been decided to use anticoagulants, treatment is started immediately with heparin. This is given intravenously in a continuous 250 to 500 cc. saline infusion with a total dose equivalent to 50 to 100 mg. of heparin every four hours. The infusion is adjusted so that it will take twelve hours to run in. This is admittedly a cumbersome method to both the patient and the physician, but the use

of Pitkin's menstruum is precluded by expense and, in a minor way, by pain at the site of injection. We have had limited experience, thus far, in the use of Depo–Heparin. In some institutions a lapse of six to eight hours is allowed between the last night dose of heparin and the first morning dose.[23] We do not feel justified in allowing such a lapse — hence the use of continuous infusion. At the same time, after a prothrombin test is done, administration of Dicumarol is started. We have found that a dose of 300 mg. the first day, 200 mg. the second day and 100 mg. the third day is a good average one. This is by no means invariable, however, and the dosage of Dicumarol should, at all times, be guided by the daily prothrombin time. In some cases larger doses are required, and in others, smaller amounts. By the third day, it is usually possible to discontinue heparin and to maintain the anticoagulant effect with Dicumarol by keeping the prothrombin percentage between 45 and 60.

As to determination of the anticoagulant effect of heparin we have accepted the work of Jorpes[24] in which he states that it is not necessary to perform daily estimates of the coagulation time when heparin is used in doses of 50 to 100 mg. every four hours. He has shown that this amount provides adequate anticoagulant effect and is rarely, if ever, associated with bleeding. The coagulation time is determined at the onset of treatment, however, to rule out any disorder of the coagulation system. The antidote for excessive heparin effect is protamine sulfate, given intravenously in 1 per cent sterile solution in doses of 50 to 100 mg. To date we have not needed to use this antidote. The antidote for excessive Dicumarol effect is vitamin K in doses of 68 mg. intravenously every four hours and fresh whole-blood transfusions. We have had to use vitamin K in 3 cases: in 1 to control vaginal bleeding probably due to Dicumarol in a postoperative hysterectomy and in 2 other patients under Dicumarol effect to raise the prothrombin percentage so that venous interruption could be done.

How long should a patient be kept on Dicumarol? In attempting to answer this question, we have been guided solely by the patient's clinical appearance; thus, when signs of consolidation in the chest have disappeared, when pleurisy has subsided, when sputum is no longer blood-tinged and temperature, pulse and respiration have returned to normal and leg signs have subsided, the patient is allowed up. In regard to the legs we look for regression of swelling, and tenderness and absence of Homans's sign in phlebothrombosis, and almost complete regression of swelling in thrombophlebitis, keeping in mind that some of this swelling may be a sequela after increased heat and tenderness have disappeared. In such conditions as myocardial infarction and congestive heart failure, the length of bed rest is determined basically by the disease, and the anticoagulants are continued until the patient is up, when prothrombin percentage is allowed to rise to normal. The efficacy of anticoagulants in lowering the death rate and reducing the thromboembolic phenomena following myocardial infarction has been pointed out in recent reports.[25, 26]

Although we have had no experience in the use of anticoagulants in ante-partum or post-partum patients, we can readily understand the possibility of producing hemorrhage in the newborn, through transplacental transmission of Dicumarol, when the anticoagulants are given to the mother before delivery. Kraus et al.[27] have shown this to be so experimentally. On the other hand, recent reports by Adamson[28] and Jaimet[29] advocate the use of anticoagulants in early labor and even before if indications exist. There is, however, no mention in their papers of possible deleterious effects on the fetus. Allen et al.[30] state that "Dicumarol may be used safely and with benefit in the treatment and prevention of venous thrombosis following delivery." They have used it in 19 post-partum cases successfully and without untoward bleeding.

Treatment by Venous Interruption

At present the trend in general appears away from venous interruption in venous thrombosis with the increasing use of anticoagulants. This has been true in the Heywood Hospital, but we do not believe that anticoagulants are the sole answer to the problem. In our opinion, certain patients should be treated by venous interruption alone. In some cases interruption is used in conjunction with anticoagulants.

As mentioned above, the laboratory of a community hospital must be able to produce consistently accurate prothrombin determinations for the control of Dicumarol day in and day out. Until this was achieved at the Heywood Hospital, venous interruption was the procedure of choice, and from our own experience, we consider it the only method to be employed in any hospital that finds itself in a similar situation.

The indications for venous interruption are listed in Table 1. Since this part of the problem is one that always elicits discussion and argument, a few remarks about our indications are necessary.

A high incidence of venous thrombosis and thromboembolism in patients with fractured hips, major amputations or severe trauma to the lower extremities is a known fact.[31, 32] In the elderly patient we are hesitant to administer anticoagulants; therefore, our prophylactic measure of choice is venous interruption. At present our orthopedic surgeons are divided in opinion. Some prefer to handle the problem as it arises rather than to carry out routine interruptions. One of them favors prophylactic venous interruption. In view of the statistics, the latter procedure appears the safer method.[33] In elderly patients undergoing extensive

gastrointestinal operations, we believe that prophylactic venous interruption is indicated, particularly if they are to have stage operations. In most of the cases in which this prophylaxis has been neglected, it has become necessary in the early postoperative period.

Because of its slowness of action Dicumarol is never used as an emergency treatment in the patient with pulmonary infarction when heparin is not immediately available. Venous interruption then becomes the treatment of choice, as it does in the occasional patient who suffers repeated emboli with an adequate anticoagulant level. It goes without saying that the patient with active bleeding, or one in whom there is the possibility of bleeding, should have his veins treated surgically.

Although we have not had occasion to do so, we should use venous interruption in the ante-partum patient with venous thrombosis, since there is some evidence that the anticoagulants pass through the placenta and are transmitted to the fetus.[27]

Finally, we believe that any patient who suffers a massive pulmonary embolism should be treated by immediate venous interruption with the simultaneous administration of anticoagulants. In our opinion, this is an emergency procedure.

Authoritative discussion of the technic of femoral-vein interruption has already been published in excellent articles by Allen,[32, 33] Linton[34] and Edwards and Robuck.[35] We can add nothing but certain observations that we have made while interrupting the veins of 46 patients in a small community hospital. The operation is neither difficult nor dangerous, requiring only care and average surgical skill in its performance. As emphasized by the authors cited above, it is our practice to interrupt and explore these veins, not merely to tie them. Mere ligation of veins may do as well in the treatment of the immediate condition, but such a procedure not only leaves the clot in situ but also offers little protection against recanalization of the vein. Should such a vein recanalize, it does so in the presence of a valvular system that is impaired, and a chronically swollen leg may be the result.

The operation can be easily done under local anesthesia, and we have never hesitated to operate because the patient is too ill. In fact, immediate improvement has occurred, dramatically, in a sufficient number of cases to be more than mere coincidence.

We always open the vein and explore it for clot. At first, we were rather apprehensive about this because we feared that we might dislodge a clot and cause a fatal embolism on the operating table. We have never seen this happen; in fact, we have been impressed with the amount of actual back pressure in the vein. In other words, when the vein is opened and the clot is sucked out of the proximal end, there has always been a marked gush of blood with more than expected force. We therefore think the danger of loosening a fatal embolism during operation has been overemphasized.

Because there is always some doubt where to ligate, for our own purposes, we go according to the following rules. On the premise that the apparently unaffected leg often harbors the unsuspected thrombus, bilateral femoral-vein interruption is always done. This was our procedure in all 46 patients on whom the operation was done in the Heywood Hospital. In spite of the emphasis in the literature on this subject, unilateral femoral-vein interruption is occasionally mentioned. Such a procedure not only is inadequate but also may compromise the patient with a more dangerous situation than he had before operation.

It is our usual procedure to interrupt the superficial femoral vein unless a clot is found to extend into the common femoral vein, whereupon the common femoral vein is explored, the clot removed and the vein interrupted. One can easily suck down 2.5 to 5 cm. of clot from the common femoral vein above the point of the incision. However, surgical obstruction of the common femoral vein is to be avoided whenever possible since it seriously impairs the collateral circulation.[36]

The operation of vena-cava ligation is not technically difficult, but because it may produce serious disabling postoperative sequelae, it is our practice to consult a vascular surgeon in these cases. Ligation of the vena cava was performed in 2 cases because of repeated emboli after adequate use of anticoagulants and femoral-vein interruption, and both patients were operated on by specialists in this field. So far as possible, we shall follow this method of handling such cases in the future.

We have not used sympathetic block in this hospital for the treatment of venous thrombosis, our experience having been confined to its uses in arterial disease. We have used it on occasions in other hospitals in the treatment of thrombophlebitis, where it has a distinct value, and we plan to use it in the future as an adjunct to our present program.

We have used papaverine for vasodilation in these cases, with moderate benefit. With the present development of newer and more efficient vasodilators,[37] possibly these will take the place of papaverine and sympathetic block for the relief of reflex spasm in the collateral circulation.

Case Reports

Case 1. Mrs. H. S. (H.H.M.H.73009), a 64-year-old woman, was admitted to the hospital on September 4, 1948, in frank congestive failure. In addition to the usual measures for heart failure, Dicumarol was administered when signs of phlebothrombosis appeared in the left leg. Through lack of knowledge of the method of the test used at this hospital (the viper-venom technic as described above), the prothrombin level was lowered to 24 per cent as had been the custom at a previous institution. She was discharged on September 28, when the prothrombin level was 80 per cent. On October 1, however, gross hematuria developed, and the patient was readmitted. Her course on the second admission was

rapidly fatal. Hematuria persisted in spite of vitamin K in 72-mg. doses intravenously every 4 hours and whole-blood transfusions. The nonprotein nitrogen rose steadily. She became comatose and died on October 6. At no time after the first discharge was the prothrombin level below 80 per cent, and during the second admission it was 80 to 100 per cent. Autopsy showed gross bleeding from the genitourinary tract but little or no signs of active bleeding elsewhere.

Although autopsy findings do not conclusively substantiate the clinical impression of Dicumarol poisoning, this case points up an important lesson. This patient was given an excessive amount of Dicumarol because of lack of complete understanding of the prothrombin test used on the part of the attending physician. Whether or not she died of Dicumarol poisoning is not the issue but rather the lack of liaison between the clinician and laboratory. At present we believe that the prothrombin test as done at the Henry Heywood Memorial Hospital is adequate and reliable.

Case 2. Miss B. Y. (H. H. M. H. 75153), a 49-year-old woman, was readmitted to the hospital on April 9, 1949, 1 month after a hysterectomy, because of a dry hacking cough without hemoptysis of 2 days' duration and severe, knife-like pain, sharply limiting respiration in the right lower portion of the chest and radiating to the right shoulder. There were signs of consolidation over the right lower lobe, but no friction rub was heard. X-ray examination of the chest showed an area of increased density of the right lower lobe. There were no leg signs. Administration of heparin was started immediately (200 mg. in 500 cc. of physiologic saline solution). During this infusion she had a chill, after which the temperature rose to 103°F. The urine at this time showed a high trace of albumin, and the sediment was loaded with red blood cells. For fear that she had or might have extrarenal azotemia, it was decided that further therapy with anticoagulants was contraindicated. Accordingly, bilateral femoral-vein interruption was done, with removal of a large clot from the left superficial femoral vein. On the following day the urine again showed a high trace of albumin, with many red blood cells in the sediment, and the nonprotein nitrogen was 48 mg. per 100 cc. On April 11 administration of Dicumarol was resumed, after the urine had begun to clear and the nonprotein nitrogen decreased. The remainder of the hospital course was uneventful.

This case aptly illustrates the complementary use of venous interruption and anticoagulants in the treatment of venous thrombosis with pulmonary infarction. It also points out that contraindications to anticoagulants are not necessarily well established conditions but may arise quite quickly, as in this case, in the form of transient renal involvement.

Case 3. Mrs. M. G. (H. H. M. H. 74306), a 47-year-old woman, was given Dicumarol 3 days after hysterectomy, when a superficial phlebitis of the right leg and calf tenderness with Homans's sign in the left leg developed. On the 8th day of anticoagulant therapy, when the prothrombin was 43 per cent, supposedly in the therapeutic range, bleeding from the vagina began. Because it was believed that the relation between Dicumarol and the bleeding was one of cause and effect, in spite of a therapeutic prothrombin level, 68 mg. of vitamin K was given intravenously. On the following day the prothrombin was 73 per cent, but she passed a large clot. Two more doses of vitamin K of 68 mg. were given intravenously, 4 hours apart. After this the prothrombin rose to 90 per cent, and there was no further sign of bleeding. The signs in the left leg had subsided, and there was only minimal tenderness in the right leg.

This case is presented for two reasons: to illustrate the use of anticoagulants as prophylaxis against phlebothrombosis in one leg when the opposite leg is the site of thrombophlebitis; and to demonstrate the use of vitamin K in raising the prothrombin level when it is believed that bleeding may be due to an excessive Dicumarol effect.

Case 4. Mrs. M. D. (H. H. M. H. 72493), a 59-year-old woman, had a panhysterectomy for adenocarcinoma of the uterus on July 13, 1948. She was admitted to the Heywood Hospital on August 15, with a 4-day history of substernal chest pain that had become pleuritic. On the morning of admission, she coughed up some bright-red blood. Further history revealed that she had been having shooting pains in the leg, with some swelling of the ankles. These signs, however, had been present before operation. Physical examination revealed a dyspneic, cyanotic woman with engorged cervical veins and signs of consolidation over the right lower lobe and with crepitant rales over the left lower lobe. The heart was enlarged, the area of cardiac dullness extending to the left, with a rate of 120, and a gallop rhythm was present. There was moderate pretibial edema, but no calf tenderness or Homans's sign. It was believed that she had had a pulmonary embolism with infarction and that when admitted she was in congestive failure. The congestive failure was treated in the usual manner, and she responded. There were no further episodes of pulmonary infarction even though she was treated conservatively from this aspect since anticoagulants could not be administered, for lack of laboratory control, and since femoral-vein interruption was not done because it was believed that the iliac vessels were the source of the embolus. She was discharged on September 5. Two days later she was admitted because of a bout of severe, knife-like pain in the left side of the chest that markedly limited respiration and was associated with a cough productive of brownish sputum. There were signs of consolidation over both lobes. Nasal administration of oxygen and papaverine (0.06 gm. every 4 hours) intramuscularly, as well as penicillin, were given. At this time we believed that we could administer anticoagulants with good laboratory control. Therefore, Dicumarol therapy was started; a therapeutic level was achieved and maintained, in spite of which the patient had two more bouts of severe pleurisy with hemoptysis and with a drop in blood pressure and rapid pulse. The findings in the chest were the same except for the addition of a loud friction rub over the right lower lobe. By September 17 the friction rub had disappeared. At no time were there signs in the legs, and it was believed that the emboli were arising from the pelvic veins. It soon became apparent that she could not be kept on anticoagulants indefinitely; therefore, after consultation with a vascular surgeon, it was decided to interrupt the femoral veins. Vitamin K was given intravenously to return the prothrombin time to normal. Femoral-vein interruption (with removal of clots on both sides) was done on October 7. On the following day she had a recurrence of severe pain in the left side of the chest with a drop in blood pressure, rapid pulse, profuse perspiration and cyanosis. Examination of the chest showed signs of consolidation over the left lower lobe and a friction rub. Dicumarol was given again, and the prothrombin percentage lowered to the therapeutic level. On October 12 the friction rub was no longer heard, and from then on she made a rapid recovery. On October 19 she was allowed up, and several days later Dicumarol was discontinued. She was discharged on October 27. She had been seen several times since and is apparently well.

This complicated case illustrates very well how the two methods of treatment for venous thrombosis and pulmonary embolism can be used together to the ultimate benefit of the patient. During the first admission for pulmonary embolism, treatment was conservative since we dared not use anticoagulants for lack of laboratory control, and femoral-vein interruption was not done since it was believed

that the source of emboli was in the pelvic veins. She recovered from that particular episode, only to succumb to a recurrence of pulmonary emboli. Conservative treatment was obviously inadequate. The second bout of pulmonary embolism was treated with Dicumarol since laboratory test was now available, but in spite of adequate anticoagulation she suffered a third embolism. At this time, as indicated above, femoral-vein interruption was done after prothrombin per cent was returned to normal with Vitamin K. Subsequently, the fourth embolism occurred, and Dicumarol was started again and maintained until the patient was out of bed. Although it is hard to prove, it is difficult to conceive that this patient would have survived with either method of treatment used alone. The final successful result seems directly attributable to the use of both methods in a complementary fashion.

Case 5. E. S. (H. H. M. H. 69171), a 61-year-old man, was admitted to the hospital on October 23, 1947, with a diagnosis of carcinoma of the rectum and bilateral inguinal hernias. On October 28 a one-stage abdominoperineal resection was performed. Ambulation was started 36 hours postoperatively with leg exercises. Convalescence was normal until November 7, when a right-sided strangulated hernia developed that was reduced under spinal anesthesia. Four days later there were signs of pulmonary infarction with cyanosis sufficient to require an oxygen tent. He also exhibited right-calf tenderness. Under local anesthesia, bilateral femoral-vein interruption was done. On the right side, a clot was removed from the femoral vein, and the common femoral vein was interrupted. Superficial femoral-vein interruption was done on the left side. Further convalescence was uneventful.

The patient's stated age was sixty-one but in physical appearance he was ten to fifteen years older. In retrospect, we believe that a prophylactic femoral-vein interruption should have been done at the first operation or, if not then, most certainly at the time of the strangulated hernia.

Case 6. Mrs. D. S. (H. H. M. H. 73871), a 74-year-old woman, was admitted to the hospital with a diagnosis of carcinoma of the rectum. The history was suggestive of phlebothrombosis many years previously at childbirth. On December 22, 1948, a first-stage Lahey-type resection and bilateral superficial femoral-vein interruption were done. The second stage was done on January 10, 1949. Convalescence was essentially uneventful, with minimal leg discomfort. There were no pulmonary complications. The patient has no trouble with her legs at present other than minimal swelling.

This case is presented to illustrate what we consider the proper handling of a person in this age group having stage operations. At no time did she show signs suggestive of thromboembolism, a complication that admittedly would have been extremely likely in this case.

Case 7. Mrs. D. S. (H. H. M. H. 75694), a 46-year-old woman, was admitted to the hospital on May 23, 1949, with a diagnosis of acute thrombophlebitis of the right leg. An enlarged, hard uterus was found on physical examination. The patient gave a history of irregular vaginal bleeding 5 months before admission and regular periods subsequently. She was given papaverine and Dicumarol. The therapeutic level was approached in 48 hours, and bleeding from the vagina began. Seventy-two milligrams of vitamin K was given intravenously, and bilateral superficial femoral-vein interruption was done. Bleeding was controlled as long as the patient remained in bed. On June 2 a transfusion was given, and panhysterectomy done without event. The pathological diagnosis was adenomyosis of the uterus. Convalescence was uneventful. The patient was discharged on June 9. The swelling of the right leg rapidly cleared.

Anticoagulant therapy was started in this case, but it quickly became evident that such treatment was contraindicated and femoral-vein interruption then became the treatment of choice.

Case 8. Mrs. F. N. (H. H. M. H. 68287), a 29-year-old woman, was admitted to the hospital on August 3, 1947, with acute cholecystitis. Cholecystectomy was done. On the 1st postoperative day, marked abdominal distention developed and was quickly and easily controlled with intestinal intubation. The drain was removed on August 10, and on the following day, the patient complained of severe right-upper-quadrant pain. The legs were checked daily from the day of operation; no signs of phlebothrombosis were elicited. X-ray examination showed slight elevation of the right leaf of the diaphragm. A spiking temperature with intermittent right-upper-quadrant pain continued. Examination of the legs two or three times a day elicited no positive signs. There was no evidence of wound infection and no apparent pocketing. Examination of the chest was negative except for a few slight rales at the right base. The course continued unchanged until August 17, when it was decided to do a venous interruption in spite of the absence of signs. Coincident with this decision, for the first time, calf tenderness was elicited in the right leg. Operation was done under local anesthesia. The right femoral vein was found to be filled with clot, which was removed, and the common femoral vein was interrupted. On the left, the superficial femoral vein was interrupted. The following day, the patient was dramatically improved and she was discharged on August 26. Swelling of the extremities continued for about 8 weeks after discharge but was not disabling. The patient now has no swelling of the extremities and has been well since discharge.

This case illustrates two points. First, in spite of repeated examinations, the diagnosis was not made until late in the course of the disease, and only after a pulmonary embolism had occurred. Secondly, this patient improved dramatically after venous interruption, and we have seen such an improvement sufficiently often to regard it as more than mere coincidence.

Discussion

Although the disturbing thought occasionally arises that the whole problem of venous thrombosis and pulmonary embolism has been emphasized out of proportion to its actual importance, an evaluation of the statistics in the Henry Heywood Memorial Hospital, accruing from conservative treatment and those resulting from active treatment makes the latter course mandatory. From June, 1944, to January, 1947, there were 11 deaths from pulmonary embolism among 9438 admissions. From January, 1947, to June, 1949, a period in which the staff practiced an increasingly active approach to the problem, there were 5 deaths from pulmonary embolism among 9915 admissions. In none of these deaths were anticoagulants used or venous interruptions performed, and in retrospect, a review of our records leads us to believe that, in the majority of the cases, there was ample opportunity

to do so. By contrast, during the later period, we treated 80 patients with venous thrombosis or its complications by anticoagulants or venous interruption or both. In this group, there were no deaths from pulmonary embolism. Such figures testify that our attitude is hardly one of overemphasis.

Prevention of pulmonary embolism lies in the early diagnosis of venous thrombosis, which centers around the concept that phlebothrombosis and thrombophlebitis differ, clinically and pathologically. The former is the dangerous source of emboli; the latter is a much less dangerous source. The two methods used for treatment — anticoagulants and venous interruption — should be considered as being complementary to each other. The trend seems to be in the increasing use of the anticoagulants and perhaps justifiably so, since it is a more physiologic method, attacking as it does the basic lesion throughout the vascular system, rather than only the venous system of the legs. In addition, the use of anticoagulants minimizes the incidence of post-phlebitic or post-thrombotic sequelae[38, 39] — that is, edema, ulceration and infection. These sequelae are believed to be due to reflex arteriolar spasm and chronic venous insufficiency.[3, 38, 40] Sympathetic block is an effective means of combating reflex arteriolar spasm,[3, 40] and reports on the use of the newer vasodilators, such as Priscol, appear encouraging.

We do not believe, at present, that the anticoagulants will render obsolete the use of femoral-vein interruption, for we have been faced on numerous occasions with cases in which a contraindication to anticoagulants exists or arises. By this we do not mean the overt contraindications, such as active bleeding and blood dyscrasias, but rather sudden complications such as temporary renal involvement, as illustrated in 1 of our cases, or unexplained resistance to anticoagulants. On the other hand, we recognize no contraindications to venous interruption. This is a point well worth remembering. In massive infarction, the patient deserves the benefit of both anticoagulants and venous interruption.

The anticoagulants, when given under adequate and rigid laboratory control, can be administered safely. Under the circumstances hemorrhage, as a complication, is uncommon. When it does occur, effective antidotes are available. It is possible that we could extend the use of anticoagulants to older patients, but we do not believe that it is yet safe for us to do so. There is, however, a need for an anticoagulant that is easier of administration and control than either heparin or Dicumarol. The expense of repeated prothrombin tests or repeated injections of heparin is a major consideration.

Finally, all investigators recognize that the complete answer to this problem will be forthcoming only after much more study. In the meantime, it has been our purpose to clarify to some degree our approach to treatment.

Summary

The incidence of venous thrombosis and its complications in a 125-bed hospital is of sufficient magnitude to require some logical guide to treatment.

A plan of attack that, in the light of present-day knowledge, seems rational and is adaptable to our hospital situation is presented.

References

1. Getting, V. A., Rubenstein, A. D., and Ryder, C. F. Hospital and health facilities in Massachusetts: Massachusetts State Plan for administration of Public Law 725 (Hospital Survey and Construction Act). *New Eng. J. Med.* **239**:1028-1032, 1948.
2. Ochsner, A., and DeBakey, M. E. Thrombophlebitis and phlebothrombosis. *South. Surgeon* **8**:269-290, 1939.
3. Ochsner, A. Venous thrombosis. *J. A. M. A.* **132**:827-833, 1946.
4. Homans, J. Venous thrombosis and pulmonary embolism. *New Eng. J. Med.* **236**:196-201, 1947.
5. Mendelsohn, S. N. Early walking after major gynecologic surgery. *Am. J. Surg.* **71**:614-619, 1946.
6. Canavarro, K. Early postoperative ambulation. *Ann. Surg.* **124**: 180, 1946.
7. Burch, J. C., and Fisher, H. C. Early ambulation in abdominal surgery. *Ann. Surg.* **124**:791-798, 1946.
8. Keyes, T. F. Observations on early postoperative rising. *Am. J. Surg.* **74**:192, 1947.
9. Albright, H. L. Thrombophlebitis: problem of treatment. *New Eng. J. Med.* **236**:928-933, 1947.
10. Borgström, S. Value of postoperative dicoumarin prophylaxis at "early rising." *Acta chir. Scandinav.* **96**:47-61, 1947.
11. Strömbeck, J. P. Attempt to evaluate different modern methods for prevention and treatment of thromboembolism. *Acta chir. Scandinav.* **97**:113-134, 1949.
12. Sulamaa, M., and Penth, A. On thrombo-embolia diagnostics in practice and on need of anticoagulant prophylaxis. *Acta. chir. Scandinav.* **98**:130-141, 1949.
13. Blodgett, J. B., and Beatie, E. J. Early postoperative rising: statistical study of hospital complications. *Surg., Gynec. & Obst.* **82**: 485-489, 1946.
14. Allen, A. W., Linton, R. R., and Donaldson, G. A. Venous thrombosis and pulmonary embolism. *J. A. M. A.* **133**:1268-1276, 1947.
15. Powers, J. H. Postoperative thromboembolism: some remarks on influence of early ambulation. *Am. J. Med.* **3**:224-230, 1947.
16. Blodgett, J. B. Early ambulation following surgical procedures. *Bull. New York Acad. Med.* **25**:176-184, 1949.
17. Carlotti, J., Hardy, I. B., Linton, R. R., and White, P. D. Pulmonary embolism in medical patients, comparison of incidence, diagnosis and effect of treatment in two hundred and seventy-three cases at Massachusetts General Hospital in two five-year periods (1936 to 1940 and 1941 to 1945 inclusive). *J. A. M. A.* **134**:1447-1452, 1947.
18. Allen, E. V. Clinical use of anticoagulants: report of treatment with dicumarol in 1,686 postoperative cases. *J. A. M. A.* **134**:323-329, 1947.
19. Loewe, L. Anticoagulation therapy with heparin/Pitkin menstruum in thromboembolic disease. *Am. J. Med.* **3**:447-467, 1947.
20. Page, R. C., and Russell, H. K. Prothrombin estimation using Russell viper venom: simple modification of Quick's method. *J. Lab. & Clin. Med.* **26**:1366-1370, 1941.
21. Page, R. C., and de Beer, E. J. Prothrombin studies using Russell viper venom: interbatch variations of venom. *J. Lab. & Clin. Med.* **28**:912-915, 1943.
22. Sparling, H. J. Personal communication.
23. Starr, A. Femoral vein ligation versus heparin therapy of deep venous thrombosis. *Bull. New Eng. M. Center* **9**:206-210, 1947.
24. Jorpes, J. E. Origin and physiology of heparin: specific therapy in thrombosis. *Ann. Int. Med.* **27**:361-370, 1947.
25. Wright, I. S., Marple, C. D., and Beck, D. F. Report of Committee for the Evaluation of Anticoagulants in the Treatment of Coronary Thrombosis with Myocardial Infarction. *Am. Heart J.* **36**:801-815, 1948.
26. Wright, I. S. Experience with dicumarol (3,3′ methylene-bis-[4 hydroxycoumarin]) in treatment of coronary thrombosis with myocardial infarction. *Am. Heart J.* **32**:20-31, 1946.
27. Kraus, A. P., Perlow, S., and Singer, K. Danger of dicumarol treatment in pregnancy. *J. A. M. A.* **139**:758-762, 1949.
28. Adamson, D. L., Weaver, R. T., and Jaimet, C. H. New view on use of dicumarol in pregnant patient. *Canad. M. A. J.* **61**:6-10, 1949.
29. Jaimet, C. H. Anticoagulant therapy. *Canad. M. A. J.* **61**:10-13, 1949.
30. Allen, E. V., Hines, E. A., Jr., Kvale, W. F., and Barker, N. W. Use of dicumarol as anticoagulant: experience in 2,307 cases. *Ann. Int. Med.* **27**:371-381, 1947.

31. Veal, J. R. Prevention of pulmonary complications following thigh amputations by high ligation of femoral vein. *J. A. M. A.* **121**: 240-244, 1943.
32. Allen, A. W. Interruption of deep veins of lower extremities in prevention and treatment of thrombosis and embolism. *Surg., Gynec. & Obst.* **84**:519-527, 1947.
33. *Idem.* Present evaluation of prophylaxis and treatment of venous thrombosis and pulmonary embolism. *Surgery* **26**:1-7, 1949.
34. Linton, R. R. Venous interruption in thromboembolic disease. *Surgery* **19**:434-436, 1946.
35. Edwards, E. A., and Robuck, J. D., Jr. Applied anatomy of femoral vein and its tributaries. *Surg., Gynec. & Obst.* **85**:547-557, 1947.
36. Homans, J. Management of recovery from venous thrombosis in lower limbs. *Surgery* **26**:8-15, 1949.
37. Rogers, M. Use of sympatholytic drug (priscol): from point of view of general surgical practitioner. *J. A. M. A.* **140**:272-276, 1949.
38. Linton, R. R., and Hardy, I. B., Jr. Post-thrombotic sequelae of lower extremity: treatment by superficial femoral vein interruption and stripping of saphenous vein (preliminary report). *S. Clin. North America* **27**:1171-1177, 1947.
39. Levan, J. B. Dicoumarol therapy. *Ann. Int. Med.* **25**:941-949, 1946.
40. Ochsner, A., and DeBakey, M. Postphlebitic sequelae. *J. A. M. A.* **139**:423-429, 1949.

SUBTOTAL ESOPHAGECTOMY WITH CERVICAL ESOPHAGOGASTROSTOMY*

Report of a Case

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SINCE the publication of three separate reports[1-3] of successful resection of the esophagus with anastomosis in the neck, it seems reasonable to conclude that this procedure has been established as a feasible maneuver. These patients were sixty-eight, fifty-nine and sixty-one years of age, and all stood the operation without untoward complications. One wonders if now is not the time to re-evaluate the surgical approach to carcinoma of the esophagus and to perform this radical resection more frequently.

CASE REPORT

A 72-year-old widow entered the hospital on January 20, 1949, with a complaint of not being able to swallow anything but liquids for 2 months and regurgitation of liquids for 2 days. She had lost an unknown amount of weight. X-ray films of the esophagus after a barium swallow had shown an obstructing lesion at the upper margin of the arch of the aorta.

Physical examination revealed an alert woman who did not appear as old as her stated age. The blood pressure was 160/90. There were numerous dilated veins in the saphenous systems of both legs. X-ray films of the chest and an electrocardiogram were within normal limits. Examination of the blood disclosed a red-cell count of 4,320,000, with a hemoglobin of 11.6 gm. per 100 cc. The nonprotein nitrogen was 38 mg., and the serum protein 6.35 gm. per 100 cc. The chloride was 510 milliequiv. per liter, and the carbon dioxide 50 vol. per cent. The urine showed a specific gravity of 1.020, a faint trace of albumin and no sugar. Examination of the sediment was negative.

Esophagoscopy on January 22 revealed an obstructing lesion 11 cm. from the upper incisor teeth. Biopsy was taken and reported on January 23 as epidermoid carcinoma (Grade II).

On January 24, under intratracheal ether anesthesia, an incision was made over the eighth rib, which was removed subperiosteally. The left lower lobe was adherent over a considerable area, with moderately dense adhesions, which were freed. After incision of the mediastinal pleura above the arch of the aorta, the tumor was felt well above the arch and extending down behind it. It was thought to be removable. Sections of the seventh, sixth and fifth ribs were removed posteriorly to allow greater exposure. The esophagus was then freed throughout its extent, some difficulty being encountered in dissecting the tumor-bearing area from the aortic arch. This dissection was facilitated by division of the upper two intercostal arteries and rotation of the aorta forward. The thoracic duct was encountered and, because of trauma to it, was divided and ligated at both ends. A suspicious lymph node was felt in the lower periesophageal tissues. The phrenic nerve was crushed. The diaphragm was divided, and the liver and abdominal cavity were found free of disease. The esophagus was then separated from the stomach by division of the upper stomach below the junction with the esophagus. The stomach was closed with a running suture of fine-chromic catgut reinforced with a layer of interrupted silk sutures. The stomach was then freed of its attachments by division of the branch vessels along the greater and lesser curvatures, the epiploic arteries and veins being preserved. The left gastric artery was ligated at its origin. The spleen was not disturbed. The stomach could then be lifted to the apex of the chest. The esophagus was brought anterior to the arch and freed up additionally in the superior mediastinum. The right pleural cavity was entered in this dissection, and to prevent collapse of the right lung, both lungs were forcibly expanded and a wet-gauze pack placed over the rent in the pleura until the chest had been closed. Palpation of the tumor at this point indicated that it would be unwise to attempt a thoracic anastomosis since the uppermost limits of the mass were less than 2.5 cm. from the thoracic outlet. The stomach was sutured high in the chest to the surrounding thoracic and mediastinal pleura. The diaphragm was closed around the pylorus, and the lung reinflated. A Foley catheter was brought out through the ninth interspace for drainage, and the wound was closed.

The patient was then turned on her back, and an incision made along the anterior border of the sternomastoid muscle, continuing down over the sternoclavicular joint. The attachments of the sternomastoid and major pectoral muscles to the inner half of the clavicle were divided, and the clavicle separated from the sternum. The inner half of the clavicle was then removed with the Gigli saw. The inner half of the first rib was removed subperiosteally. The ribbon muscles were detached from the manubrium, and a finger inserted into the superior mediastinum and the esophagus delivered into the neck. The pleura was then opened, and the fundus of the stomach elevated into the neck with Babcock clamps. It was not found necessary to remove part of the manubrium as advised by Sweet.[4] A two-layer anastomosis was made between esophagus and stomach as high above the tumor as possible — consisting of an inner row of interrupted fine-silk sutures through all layers and an outer row of mattress sutures of silk through the esophageal muscularis and the layers of stomach down to the submucosa. The esophagus bled freely on section. The part of the stomach brought into the neck was sutured to the surrounding muscle to prevent tension on the anastomosis. The muscles were approximated to each other and to the sternum, and the skin was closed with interrupted sutures without drainage. Silk was used throughout. The patient's condition remained good throughout the entire procedure — the pulse fluctuating between 80 and 90, and the blood pressure remaining at 140/90.

Pathological report by Dr. Irving Akerson revealed two submucosal nodules, one above and one considerably below the main tumor, and a lymph node at the lower end of the

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specimen, all showing epidermoid carcinoma (Grade III). The appearance of the specimen is shown in Figure 1.

The patient was kept in an oxygen tent for 3 days postoperatively and given penicillin and streptomycin intramuscularly. An x-ray film taken with a portable machine on the morning after operation showed a 20 per cent pneumothorax on the right side. About 400 cc. of air was aspirated. The catheter was removed from the chest after 48 hours, 1000 cc. of serosanguinous fluid having been drained. The nonprotein nitrogen was 95 mg. per 100 cc. on the 3rd postoperative day, which was attributed to hemoconcentration — a result of hesitancy to administer fluids intravenously. They were given by clysis and apparently were not absorbed sufficiently. The stomach in the neck became distended with air on the 2nd day, and a Levine tube was easily inserted through the anastomosis and left in place for 48 hours, during which bile-stained material was aspirated. On the 5th day

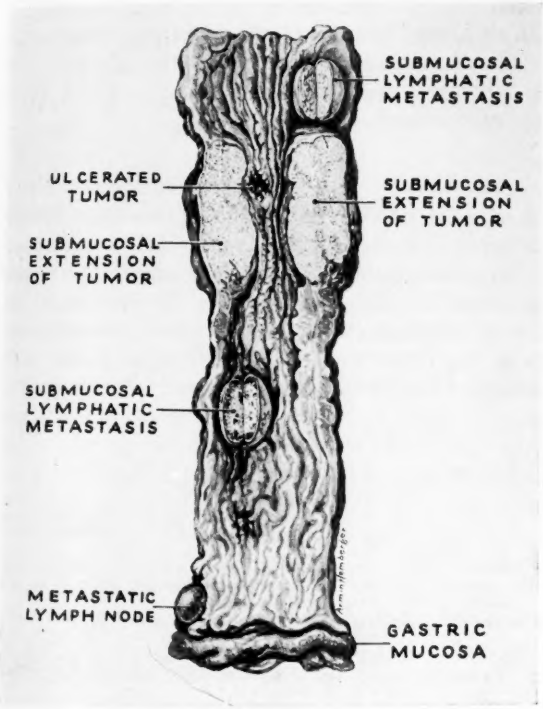

FIGURE 1. *Water-Color Drawing of the Specimen after Fixation in Formalin.*

The specimen consists of the entire thoracic esophagus, which has shrunk so that it is about 15 cm. in length — less than half its length in life.

fluids were started by mouth. On the 7th day the patient was allowed out of bed, and was eating minced foods; the nonprotein nitrogen was 45 mg. per 100 cc. On the following day the temperature rose to 101°F., and tenderness and swelling were noted in the left calf. She was given Dicumarol, and the process subsided. On the 19th day she had a coughing spell and elevation of temperature. Rales and dullness were noted in the right side of the chest, and 200 cc. of bloody fluid was aspirated from the right pleural cavity. It was concluded that the patient had a pulmonary embolus to the right lung, with hemothorax. At this time the patient's prothrombin time was 33 seconds (control, 15 seconds). Dicumarol was stopped; the patient was given a transfusion, and a bilateral femoral-vein ligation below the profunda was done. Subsequently the patient was bronchoscoped to relieve an atelectatic left lower lobe. She was discharged on the 50th postoperative day afebrile, both lungs being clear on x-ray study and fully expanded. X-ray film before discharge showed barium to flow freely through the anastomosis (Fig. 2), and there was no delay in emptying of the stomach.

The patient has had no difficulty in swallowing any kind of food. She has had to be urged to eat, however, particularly during her first 2 months at home. There has been a moderate amount of edema of the legs. Two transfusions, iron, liver and vitamins have stimulated her appetite so that in the past month she has gained 8 pounds in weight. She does her

FIGURE 2. *X-Ray Film Taken after Operation with the Patient Swallowing.*

The arrow is in the jugular notch. The divided clavicle and first rib are evident. The esophagus is the upper inch of the barium-filled gut; the anastomosis is noted at the junction with the stomach pouch identified by longitudinal striations.

own housework and is leading approximately the same life she led before being taken sick. It is now 13 months postoperatively.*

DISCUSSION

The feature of special interest in this case is the submucosal lymphatic spread, both upward and downward, from the small ulcerating tumor. Reference to the drawing of the fixed specimen (Fig. 1) will aid in visualizing the relations. The esophagus has a great tendency to shrink in its long axis after removal and fixation, so that here one sees a specimen that in life measured approximately 35 cm. shrunk to less than half that length. Also visible is the upward extension of the tumor, about 0.5 by 1 cm., which was freely movable and palpable about 1 cm. above the primary tumor mass. The downward extension was halfway between the lower margin of tumor and the stomach and actually was not discovered until the specimen was opened.

*The last follow-up examination was on February 23, 1950, at which time the patient's condition was good.

It is usual for growths in the esophagus to extend higher than the point of obstruction as demonstrated in barium studies, and for this reason it is agreed that esophagoscopy gives more accurate information for planning operative attack. The pathological description of the specimen in Nissen's[3] case is of interest:

> One and one half cm. from one end there is a firm elevated nodular submucosal area which tapers into a constricted portion. This constriction is 4 cm. from one end. Six cm. from this end the mucosa is replaced by yellow-tan, firm, papillary-like tissue which is continuous with the submucosal area described above.

It has been known that carcinoma of the esophagus has a tendency to spread by way of the intrinsic lymphatics. In a review of 108 autopsied cases at the Royal Cancer Hospital Raven[5] states, "Submucous spread in the wall of the esophagus is of important clinical significance. There may be local invasion of the wall well beyond the obvious edge of the carcinoma." At least 2 cases have been reported in which a secondary operation was necessary because of persistent tumor at the site of anastomosis, and there have been others, undoubtedly, with recurrences at this point. It is interesting to note in passing that 42.5 per cent of autopsied cases in Raven's series showed no evidence of metastases.

The history of cancer surgery has consistently followed a radical path, and coincident with more radical removal there has been an increasing percentage of cures. In the interests of cure, the object that must always be kept in mind, it therefore seems feasible to consider subtotal esophagectomy with cervical anastomosis for carcinoma of the esophagus occurring anywhere above the lower fourth, intrathoracic anastomosis being reserved for low lesions with considerable healthy tissue above them to allow an adequate resection and a safe anastomosis.

There are several features that lend support to the advisability of subtotal resection. The foremost is the fact that the blood supply of the cervical segment of the esophagus from the inferior thyroid artery is intact and abundant. It is gratifying to see pink, healthy gut of unquestionable viability when an anastomosis is made. Secondly, the anastomosis is made directly under the surgeon's eyes and at the surface rather than in the depths of the wound. Thirdly, the anastomosis is done with the patient on his back, and with both lungs expanded, favoring the general well-being of the patient during the closing period of the operation. In addition, a leak at an anastomosis in the neck would probably be less hazardous than a leak in the thorax and mediastinum, and structures could be approached by direct surgical attack in the neck with more impunity than in the chest, as pointed out by Sweet.[4]

In all 3 cases referred to above[1-3] the operation was performed without resection of the clavicle and first rib, and these maneuvers seem to be unnecessary in the average case.

Summary

A case of carcinoma of the upper third of the esophagus resected with a cervical anastomosis is presented. Submucosal spread above the tumor was noted in the specimen. It is suggested that subtotal esophagectomy should have more extended use in the treatment of lesions located lower than the upper fourth of the esophagus.

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References

1. Wylie, R. H., and Frazell, E. L. Cervical esophagogastric anastomosis following subtotal resection of esophagus for carcinoma. *Ann. Surg.* **130**:1-8, 1949.
2. Brewer, L. A., III. One stage resection of carcinoma of cervical esophagus with subpharyngeal esophagogastrostomy: report of successful case. *Ann. Surg.* **130**:9-20, 1949.
3. Nissen, R. Cervical esophagogastrostomy following resection of supra-aortic carcinoma of esophagus. *Ann. Surg.* **130**:21-30, 1949.
4. Sweet, R. H. Carcinoma of superior mediastinal segment of esophagus: technique for resection with restoration of continuity of alimentary canal. *Surgery* **24**:929-938, 1948.
5. Raven, R. W. Carcinoma of oesophagus: clinico-pathological study. *Brit. J. Surg.* **36**:70-73, 1948.

MEDICAL PROGRESS

PROCTOLOGY (Concluded)

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BOSTON

UNUSUAL PROCTOLOGIC CASES

Burt[17] reports an unusual occurrence in the case of a woman in whom he had excised a presumed blind fistulous tract extending upward from the mucocutaneous junction in the left anterior quadrant of the rectal wall. This woman had had pain in this area for a year prior to operation and had also had two previous injections of oil into some internal hemorrhoids.

At operation an area of tissue 6 by 4 by 2 cm. was excised, including the linear area of induration thought to be a fistulous tract. Two days later the patient had a temperature of 103°F., and on the third day considerable abdominal distention and tenderness and was acutely ill. Subcutaneous emphysema extended over the whole left portion of the abdomen and lower chest.

A laparotomy for suspected peritonitis or mesenteric thrombosis disclosed gas in the subcutaneous tissues, but no free abdominal air. However, there was gas behind the peritoneum everywhere within the vision of the operator, including the mesentery of the sigmoid, the descending colon, the transverse colon and the omentum. No other disease was seen. A transfusion was given, followed by penicillin and streptomycin, and the temperature reached normal on the third day after the laparotomy; the bowels moved, and the emphysema had disappeared by the sixth day.

The pathological report from the first operation described a foreign-body granuloma with disintegrating oily material in it. Culture of the rectal wound immediately after the laparotomy showed a pure growth of *Escherichia coli*.

Burt then carried out air injection in 2 dogs along the rectal wall, under pressure, and reproduced in 1 subcutaneous emphysema and in the other extensive subperitoneal extension of the air.

Whether the gas thus diffused in this woman was a result of intrarectal gas pressure that forced the intraluminal gas out through the operative wound in the rectal wall and thence upward, or whether it was gas that was generated in the pararectal tissues by gas-producing bacteria and dissected its way upward, not having access into the bowel lumen for some reason, is problematic.

The case of an infant with congenital stenosis of the entire colon and terminal ileum is reported by Cafritz and Greenberg.[18] The child was a normal 7-lb., 6-oz.-female who vomited several times about twelve hours after birth and had passed nothing by rectum. A small tube in the rectum could be passed for only 5 to 7 cm., and a diagnosis was made by barium enema.

At operation, twenty-four hours after birth, the entire colon and terminal ileum were of lead-pencil diameter, and the small bowel above was quite distended immediately proximal to the stenosed ileum. A small persistent vitellointestinal duct extended to the umbilicus from the dilated ileum. This was tied off, and a lateral anastomosis carried out between dilated ileum and descending colon. For three days all went well, but on the fifth day a Miller–Abbott tube was inserted, and could not be made to go past the duodenum. A second operation was done on the tenth day, but the child died of peritonitis on the eighteenth day. At autopsy it was thought that the tied-off duct to the umbilicus had been in the nature of a Meckel's diverticulum. Also, it was apparent that the anastomosis of ileum to colon was a mere gesture since the colon was too narrow to be of any value in eliminating the small-bowel contents. Of interest, also, was a finding of gangrene in the duodenum, which was thought to have been due to pressure of the mercury in the bulb of the Miller–Abbott tube.

A case of complete duplication of the colon and rectum and terminal ileum in a four-and-a-half-year-old child is reported in detail by Allard, Ross and Hopkirk.[19] The complaints that brought the child to the hospital were the passage of gas per urethra, frequency, dysuria and painful bowel movements. Since birth there had been alternating periods of constipation and diarrhea, with enlargement of the abdomen when the patient was constipated. The rectourethral fistula that produced the air by urethra was of four-and-a-half months' duration and had presumably been caused by ulceration produced by a prune stone found in one of several fecaliths.

Examination on admission revealed a large abdomen and a lemon-sized mass felt by rectum, which seemed to consist of smaller hard lumps that were thought to be calculi in the bladder but later proved to be hard fecal lumps in the blind lower end of the duplication. There was feces in the urine.

The exact degree of duplication and the points of connection between the two parallel colons could

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not be determined by x-ray study, and it was only after removal of the colon that the entire picture became clear.

The surgical procedures started with a transverse colostomy. When this was opened in 48° a duplication of the colostomy loop was observed to be present. Then, after cystoscopy had demonstrated a definite urethrorectal fistula, a perineal approach was used to interrupt the fistula and close the openings into urethra and rectum. The fecaliths were removed from the blind terminal rectal duplication at the same time.

Four weeks later the abdomen was again opened, and the situation reviewed, revealing duplication of the entire colon and 12.7 cm. of terminal ileum, with two appendixes. A rectal tube was introduced to determine which section of the duplication communicated with the rectum, and the ileum was then anastomosed to this section at the sigmoid.

Two weeks later the duplicate ileum and colon were removed down to the sigmoid, 2.5 cm. above the ileal anastomosis, and the distal end of the duplicated colon brought to the skin surface. It was then possible to apply a crushing clamp to the spur between the two rectums and thus to convert it into one tube after three applications of the clamp. The stoma was then closed.

The situation was really that of a median septum in what appeared externally like a single colon, with all bowel layers complete and a single common blood supply, though the two appendixes and the bifurcating ileum connecting with the separate large-bowel tubes justify calling this a true case of duplication of the colon.

Holman[20] reports a case of hemangioma of the sigmoid in a nine-year-old girl. The child had first been seen at fifteen months of age with similar lesions of the right hand and tongue, which had been successfully treated at that time. Three years later a large hemangioma above the scapula had developed.

Passage of blood by rectum in considerable quantity over a six-week period brought the child to a hospital for treatment. Proctoscopy failed to reveal any source of the bleeding, but a barium enema showed multiple small scattered areas of opacity in the left iliac fossa that suggested to the radiologist the possibility of angioma. Operation disclosed a cavernous hemangioma, 4.5 cm. in length and 1.5 cm. in width, near the mesenteric border of the sigmoid. Resection with anastomosis resulted in an uneventful recovery, and the opaque areas seen on the x-ray film were apparently due to unusually thickened fibromuscular walls of the blood spaces. The tumor protruded somewhat into the bowel lumen, and there was a shallow ulcer on its mucosal surface.

Ladd and Gross make no mention of this lesion in their book, which confirms the fact of its rarity among the diseases of childhood.

Walker[21] had the unusual experience of seeing within a short period 2 patients, each of whom had a fecalith, about 8 cm. in diameter, in the cecum. In 1 case there was a coincident and presumably unrelated area of ileitis. Resection of this area and of the cecum was performed. In the other patient the fecalith was removed from the cecum through an incision over the mass.

Fecaliths are found in the sigmoid and rectum with reasonable frequency, but their occurrence in the cecum, where the bowel contents are normally fluid, is most unusual. The author seeks to explain these cases on the basis of a local peristaltic defect that forces the cecal contents backward instead of forward along the colon in the normal way. A later barium enema in the patient whose cecum was still present showed a considerable stasis of barium in this area.

From time to time reports appear of cases in which carcinoma is found in association with an anal fistula that was known to have existed prior to the malignant lesion and, therefore, presumably to be considered as having contributed to the development of the malignant change. McAnally and Dockerty,[22] in reviewing cases of carcinoma developing in cutaneous sinuses in cases from the Mayo Clinic, found 3 in which the carcinoma was definitely located along the sinus of an anal fistula. One of these was a squamous-cell carcinoma, and the other 2 were adenocarcinoma. They mention 2 cases reported by Lockhart-Mummery and Dukes, 7 by Rosser and 5 by David, in 3 of which lymphogranuloma of the rectum had existed for long periods. I have never seen a case of this sort, but recollect one reported from the Massachusetts General Hospital some years ago by Fitchet, and I have personally operated upon a patient with carcinoma in a pilonidal sinus.

Lott and Alexander[23] report a case of basal-cell epithelioma of the upper anal canal and refer to 8 other cases of this rare tumor as reported by 6 authors. No other cases could be found in a search of the literature. The case reported lay between the sphincters as a 1-cm. area of thickening that seemed to lie below the surface of the anal canal. Two biopsies were returned with the diagnosis of "rectal mucosa with chronic inflammation," but the third specimen, four months later, was reported as "basal-cell carcinoma, lying below the mucosa."

This lesion was excised locally and the wound well cauterized, and three months after it had healed the patient was given 5500 r of irradiation.

The usual tumor encountered at the anus is epidermoid carcinoma, which is also a fairly uncommon tumor, occurring, in my own experience, in a ratio of 1:100 cases of adenocarcinoma of the rectum.

Megacolon

Swenson and his associates[24] have carried out some interesting and apparently valuable obser-

vations in children with congenital megacolon (Hirschsprung's disease). By the use of multiple balloons, so arranged that one was in the splenic flexure, one in the descending colon and the third in the narrow rectosigmoid below the megacolon, it was possible to trace the course of peristaltic waves, since each balloon was connected to a separate sphygmograph. Whereas, in normal persons there was an uninterrupted wave from transverse colon to anus, in the subjects with megacolon the wave passed downward normally, through and including the dilated bowel, but stopped at the narrow rectosigmoid, where little or no contraction was observed.

On the theory, therefore, that the megacolon must be due, not to deficient contractile power in the dilated segment, but rather to a lack of peristalsis in the narrow rectum below it, Swenson conceived the idea of resecting, not the megacolon, but the rectum and rectosigmoid, and restoring continuity with the anus. This was done in 34 cases, with 1 postoperative death and apparent cure in the other 33. (The technic had been described in 1948.[25])

Barium-enema examinations as early as three months after operation showed the colon to be normal in size and function. It seems as though this was the most impressive advance in the explanation and treatment of congenital megacolon yet presented.

Cancer

Maes and Essrig[26] have thoroughly discussed the progress made in the removal of cancers of the colon and rectum, from the first published operation by Lisfranc in 1826 to the present time. A review of 66 published papers by leaders in this field furnishes a rich background for comment and deduction, and the authors speak also from their own experience with 70 cases.

For one thing it is interesting that over the past fifty years the pendulum has swung, in surgery of the free colon, from open end-to-end anastomosis through all sorts of different procedures to open end-to-end suture again. As their procedure of choice, in cases in which resection with suture is considered to be indicated, the authors employ a single row of interrupted Connell silk sutures, all tied on the inside, and also use proximal decompression with a Pezzar catheter several inches above the anastomosis, bringing it out through a hole in the omentum, and either through the upper end of the incision or through a separate stab wound. Clamps are applied to the bowel at a 60° angle at the points of resection to increase the size of the lumen and to ensure a good circulation to the antimesenteric border of the cut bowel ends. Division of the bowel between clamps is carried out with a knife, avoiding the use of a cautery, which may devitalize the bowel ends by heating the clamps.

Maes and Essrig consider abdominoperineal resection to be the operation best calculated to cure the largest number of patients with lesions at or below the peritoneal reflection, but they believe that above this point it is desirable to avoid colostomy if possible. They base this statement on certain recent pathological investigations, which tend to refute the dictum of Miles that the "zone of downward spread" of lymphatic drainage is equal in importance to the zones of lateral and upward spread. Many other surgeons also believe that it is not of great importance and that downward spread occurs only when lymphatics above are blocked — a condition occurring only late in the course of the disease.

All the valuable advances in the subject of electrolyte balance and preparation of the bowel with nonsoluble sulfonamides are discussed, together with the pros and cons of proximal decompression by nasal tube or by a tube in the bowel at time of operation, or not at all, and the postoperative measures of accepted value are considered.

In conclusion these authors believe that the present five-year cure rate, which is around 50 per cent, can be improved upon only if more and more patients are seen earlier and earlier, and that a surgical mortality of not over 5 per cent should be achieved by more and more surgeons if real progress is to be made in the handling of this disease.

Recently, there has been renewed interest in the possibility of doing a wide removal of a rectal cancer, and at the same time bringing the sigmoid down to the anal outlet, with preservation of the sphincter. In this country, Babcock and Bacon, of Philadelphia, have been the chief exponents of these procedures, which have been frowned upon by many other leading surgeons.

Aylett,[27] in England, has reviewed the subject and cited three conditions that he believes should exist if this type of operation is selected: the lower palpable margin of the neoplasm must be at least 7 or 8 cm. from the anal verge; there must be no evidence of extrarectal spread at time of operation; if the liver shows metastases sphincter preservation is the operation of choice. He utilizes a preliminary transverse colostomy ten days before the operation and resects the rectum at a point at least 3 cm. below the palpable lower edge of the tumor. Aylett reviews the writings of Miles, Babcock, Bacon, Lynch and Wangensteen regarding the possibilities of doing this sort of procedure. Babcock, in 1932, reported his results in doing a "pull through" operation in 80 patients, with a 50 per cent five-year cure rate. Miles, in his original teachings, had emphasized a "zone of downward spread" in the lymphatic drainage from carcinoma of the rectum, but later reports have indicated that the frequency and extent of downward spread have probably been overemphasized and that resection a relatively short distance below a tumor is probably

safe in a large number of cases. This will always remain a debatable point, however, and also the number of patients in whom a sphincter-preserving operation can be done with wide removal of potential metastatic areas, and at the same time, with preservation of enough bowel to come down to the anus, seems to be relatively small. A long, freely movable sigmoid seems to be one essential factor, and the presence of a well localized tumor at a level not too low, nor too high, seems to be another necessary criterion. The writings of Babcock and Bacon in this regard are well worth careful study, and at the same time the accepted and well established abdominoperineal removal with permanent colostomy must not be discarded without convincing reasons, if one is to follow out the established principle in cancer surgery of doing the most extensive operations for the smallest tumors.

Best and Blair[28] discussed this problem thoroughly before the American Surgical Association last April and reported a recent series of 51 cases in which sphincter preservation with anastomosis had been carried out in 76.3 per cent of the patients, whereas they had performed the classic abdominoperineal operation with abdominal colostomy in only 9, or 23.6 per cent, of the cases.

The sphincter-saving procedures fall into three groups: those done entirely through the abdomen with a very low anastomosis; those in which the extensive dissection was carried out abdominally but the resection and anastomosis were done through a perineal incision by pulling out of the tumor-bearing loop posteriorly, resection, and then anastomosis; and a few cases resected and anastomosed entirely through a perineal incision because the patients were very old or considered in poor shape for either of the other two procedures.

Some degree of fecal leakage occurred in 50 per cent of these patients, all of whom had been drained beside the coccyx, but these fistulas healed spontaneously in all but 1 patient, who died of recurrent disease six months after operation.

A compilation of the work of eight authors who had carried out studies of lymph-channel extension, and lymph-node involvement, in a total of 607 cases between 1922 and 1944, showed only 12 cases with involved nodes 1 or 2 cm. below the lower border of the primary lesion and only 5 cases with an involved node beyond 2 cm. Because of these figures, and considering the fact that cancers of the lower and middle portion of the rectum are known to offer a less favorable prognosis than those higher up, it seemed reasonable to predicate this decreased favorability on other factors than downward spread of lymphatic drainage. There is no doubt that lateral spread in an area so close to levators, sacrum, prostate and so forth is a factor in the higher recurrence rate of lesions in this area of the rectum. Gilchrist and David had reported a 74.4 per cent five-year survival in patients with low lesions who had uninvolved lymph nodes, though the rate dropped to 37.5 per cent with involved nodes. In tumors above the peritoneal reflection these figures were 90 and 51.4 per cent respectively.

Best believes that there may well be so little difference in five-year cure rate between cases with removal and those with preservation of the anal outlet that it is justifiable to offer one of the latter type of procedures when in the surgeon's judgment it is feasible to do so. Vernon David and T. E. Jones, in discussion, emphasized the necessity for wide removal as the point of primary importance and did not favor sphincter-saving attempts, whereas Dixon believed that there were cases in which it could be done though he had not had favorable experience with the Babcock–Bacon "pull through" procedure. Gilchrist made a plea for some agreement on the use of the anterior reflection of the parietal peritoneum from the rectum as the point of reference in the location of cancers rather than the number of centimeters above the anus, having in mind that very high tumors can intussuscept and be felt with the finger quite low in the rectum.

Papers of the American Proctologic Society and Section of Proctology of the Royal Society of Medicine of England

The current number (January, 1950) of the *American Journal of Surgery* is devoted exclusively to publication of the papers read at the fiftieth anniversary meeting of the American Proctologic Society held in Columbus last June.[29] The Section of Proctology of the Royal Society of Medicine of England had been invited to participate in the program, and about 10 men made the trip. Most of them presented papers, which were discussed by members of the American group. After the opening address of Harry E. Bacon, president of the American Proctologic Society, Louis J. Hirschman, of Detroit, gave an oration on the subject "First Fifty Years of Proctology." In this discourse tribute was paid to Joseph McDowell Matthews, who, with 12 others, had founded the Society at a meeting in Columbus in 1899.

Historical

A. Hedley Whyte, of Newcastle-upon-Tyne, then presented a paper on the "History of the Proctologic Section of the Royal Society of Medicine." The Royal Society in its earlier form had been founded in 1653. There followed other medical and surgical societies in Rome, Paris and Great Britain. Mr. Whyte reviewed the development of these various organizations, and the appearance of papers on proctologic topics from time to time until, in 1907, with a charter from the King, the Royal Society of Medicine was formed by the amalgamation of The Royal Medical and Chirurgical Society and eighteen other existing societies. From that

point onward there appeared in the proceedings names familiar in proctologic literature — Harrison Cripps, Swinford Edwards, Eve, Waterhouse, Lockhart-Mummery, Gordon Watson and Miles. Established as the Subsection of Proctology in 1913, the status of a full section of the Royal Society of Medicine was achieved in 1940.

Cancer

Cuthbert Dukes, pathologist to St. Marks Hospital, London, led off in a symposium on rectal cancer. He has kept a complete record since 1923 on all cases of cancer of the rectum — about 3000 in number — with full details on size and position of the tumor, location of all lymphatic metastases and so forth. He outlined in detail his method of preservation of specimens, by stretching or inflation with formalin, and each one is photographed together with a parallel drawing showing the location of glandular metastases, and these are made a part of the record. He classifies tumors morphologically as malignant adenoma, malignant papilloma, protuberant carcinoma, ulcerating carcinoma, stenosing carcinoma and atypical carcinoma — descriptions that I believe all surgeons can follow. Histologically, the lesions are classified as adenocarcinoma, colloid carcinoma and carcinoma simplex. The first two types can be subdivided as of low, medium and high malignancy, though the colloids in general have a less favorable prognosis, and carcinoma simplex, the third general type, is an undifferentiated, highly malignant tumor. Then the tumors are classified as A, B or C so far as their degree of spread is concerned, with a five-year survival rate of 80 to 90 per cent in the A group and 60 to 70 per cent in the B group. The C group he subdivides into C^{-1}, having only nearby lymph-node metastasis, and C_2, having more distant metastasis. Patients with over five nodes involved rarely live more than five years.

Charles W. Mayo then spoke on "Combined Abdominoperineal Resection," based on 1314 cases with 82 hospital deaths — a 6.2 per cent mortality. He gave tables on age and sex and grading of degree of malignancy, and reported a total five-year survival rate of 54.4 per cent.

W. B. Gabriel, of London, followed with an account of his experience with "Perineoabdominal Excision of the Rectum," which he uses in nearly all cases. He reported a series of 730 cases with a mortality, under the age of fifty, of 7.6 per cent in men and 6.8 per cent in women, whereas over the age of 50 it was 15.4 per cent in men and 7.4 per cent in women. His mortality in the last 85 cases had been only 2.35 per cent, which he attributed in part to the use of stainless-steel wire. Gabriel had a 100 per cent follow-up study, with a 50 per cent five-year survival rate.

Ronald W. Raven, of London, then discussed "Malignant Melanoma of the Rectum." He had been able to find only 3 cases in a fifteen-year period at the Royal Cancer Hospital, and quoted Ernest Miles as having seen only 3 in a series of over 1500 cases of carcinoma of the rectum. This tumor is highly malignant, and Raven recommends abdominoperineal resection followed in four weeks by bilateral block dissection of the ileoinguinal lymph nodes.

George Binkley, of New York City, presented a review of 125 cases of epidermoid carcinoma of the anus and rectum. He cited the incidence in relation to adenocarcinoma of about 3.6 per cent. Of the 125 cases, 93 were seen over five years previously. Ten of these had received the bulk of their treatment elsewhere. Four refused treatment, leaving 79 cases, of which 14 were considered inoperable. Of the remaining 65 patients 2 died after operation, and 5 were lost to follow-up study. Thus, of the 58 followed, 27 survived for five years, but 2 had recurrences. This figured out to a 29 per cent survival of the entire group and 41 per cent of the operable group. Treatment was so varied that it cannot be classified, consisting of radiation, local excision and radical resection with and without groin dissection.

Skin Complications of Ulcerative Colitis

E. C. B. Butler, of London, reported 4 cases of progressive ulceration of the skin occurring as a complication of chronic ulcerative colitis. Three were very severe, and 1 was less so. These lesions coincided with a flare-up of the disease with increased bowel activity. The lesions began as a small, raised papule, which broke down into an ulcer, and the legs were the commonest site. Early cultures were sterile, but later secondary infection revealed staphylococcal, streptococcal, proteus and pyocyaneus organisms. Butler advocated excision with a diathermy knife and the use of penicillin powder. If this condition is not treated promptly toxemia and death may occur.

Hemorrhoids

A review of the present status of the injection treatment of hemorrhoids was carried out by Terrell and Chewning, of Richmond, with a report of their experience over a ten-year period. Questionnaires revealed some diminution of enthusiasm around the country for this form of therapy. The inquiry went to 355 members of the American Proctologic Society, with the following results. Of this group, 166 believed a cure was obtainable in suitable cases. No complications were experienced by 160 proctologists, whereas 47 reported abscesses, hemorrhage, slough, infarction or allergic reactions, and 106 reported very mild reactions only. Quinine and urea hydrochloride was the most popular agent used, with phenol in oil second. Terrell and Chewning believe that complications can be avoided with care, and I agree with them. Their own results reported were very good — only 5 per cent of the cases coming to later surgery.

Pfeiffer reported his experience in the injection treatment of 784 patients with 5 per cent phenol in olive oil (or Wesson oil). These represented patients with internal hemorrhoids uncomplicated by any other conditions — and comprised 65.4 per cent of the cases. Only an occasional shallow slough was encountered, and a very small number of patients complained later of severe burning, itching and tenesmus that required operation.

Allowing for follow-up difficulties Pfeiffer thinks that about 78 per cent were cured and 22 per cent recurred. He believes that simple internal hemorrhoids can be cured, and second-degree hemorrhoids improved, but that only surgical excision, carefully done, guarantees a cure.

Anal Ducts

Kratzer presented a study of 20 patients with pararectal abscesses in which no internal fistulous opening could be found, and he sought to assign to the anal ducts from the crypts the role of conveyor of the infection into the pararectal tissues. (The probable importance of these structures in this regard was brought out by Tucker and Hellwig in 1934.)

Transplantation of Ureters

Appleby has utilized a new method in extensive resections for carcinoma of the rectum involving the bladder, prostate and vesicles and requiring transplantation of the ureters in association with removal of these structures. He has transplanted the right ureter into the cecum and the left into the sigmoid in 22 cases. In 7 cases, at autopsy, there was no evidence of any more renal involvement on the right than on the left side. Appleby has more recently transplanted both ureters into the cecum as a single-stage procedure in 3 cases. It is his opinion that the colostomy is about 30 per cent less wet by this method than when the ureters have been placed in the sigmoid. He has also found an average nonprotein nitrogen of 48 mg. per 100 cc. in the 3 cecal transplants, with colostomy, as against 34 mg. per 100 cc. in cases with sigmoidal transplants *without* colostomy. Also, the injection of 6 gm. of urea into a cecostomy, with hourly checks of blood and urinary urea, revealed that in four hours there was little or no absorption from the large bowel.

Perianal Sepsis

Christensen reported 8 cases of hidradenitis suppurativa, a fistula-like infection of the perianal apocrine glands. There was, of course, no evidence of a connection with the anal canal, and cure was obtained by surgical excision. There is, however, a tendency to recur.

Pilonidal Cyst

Harberson and Brintnall presented a new type of four-flap incision for removal of pilonidal cysts that they consider an advance in the treatment of this condition. Provided the midline has been avoided in the incision of previous abscesses, the incisions being placed laterally as far as possible, the closure of the secondary operation for excision of the cyst is greatly facilitated. Their procedure was well illustrated and discussed.

Anesthesia

Lundy, of the Mayo Clinic, presented a short paper entitled "Sacral Block Anesthesia in Proctologic Operations," outlining in detail his technic. This procedure has been carried out in 22,895 cases at the Mayo Clinic. He first used 1 per cent procaine with epinephrine but more recently a 1 per cent solution of Metycaine with epinephrine. A maximum of 30 cc. of solution is used in the sacral canal, with 10 cc. in each second sacral foramen, 3 cc. in each third foramen and 2 cc. in each fourth foramen.

Crigler reported on 1500 cases in which Pentothal Sodium had been used in minor anorectal procedures, with good effect. It is avoided in children under ten or twelve years. Adequate barbiturate medication, with Dilaudid and atropine, was used, and the average duration of anesthesia was fifty-one minutes, the operations averaging twenty minutes. There was no death in the group, and the only complications — none serious — were prolonged (one and a half to two hours) anesthesia in 88 patients, laryngospasm in 22 and venous thrombosis at the site of the injection in 9.

Anatomy

Mr. C. Naunton Morgan, of London, emphasized the importance of a knowledge of the composite longitudinal muscle of the anal canal, the external sphincter and the levator in relation to hemorrhoidectomy, fistulectomy and perineal excision of the rectum. He outlined the origin and function of these muscles in detail and their relation to the procedures mentioned.

References

17. Burt, C. A. V. Emphysematous acute abdomen and subcutaneous emphysema from intrarectal operation. *Ann. Surg.* **130**:277-280, 1949.
18. Cafritz, E. A., and Greenberg, M. Congenital microcolon: case report. *Ann. Surg.* **129**:285-288, 1949.
19. Allard, C. A., Ross, D. E., and Hopkirk, J. F. Complete duplication of large bowel treated by subtotal colectomy. *Ann. Surg.* **130**:249-252, 1949.
20. Holman, C. C. Haemangioma of sigmoid colon: report of case. *Brit. J. Surg.* **36**:210, 1948.
21. Walker, A. J. Caecal faecalith: report on two cases. *Brit. J. Surg.* **36**:55-58, 1948.
22. McAnally, A. K., and Dockerty, M. B. Carcinomas developing in chronic draining sinuses and fistulas. *Surg., Gynec. & Obst.* **88**: 87-96, 1949.
23. Lott, B. D., and Alexander, C. M. Basal cell carcinoma of anus. *Ann. Surg.* **130**:1101-1103, 1949.
24. Swenson, O., Rheinlander, H. F., and Diamond, I. Hirschsprung's disease: new concept of etiology: operative results in thirty-four patients. *New Eng. J. Med.* **241**:551-556, 1949.
25. Swenson, O., and Bill, A. H., Jr. Resection of rectum and rectosigmoid with preservation of sphincter for benign spastic lesions producing megacolon: experimental study. *Surgery* **24**:212-220, 1948.
26. Maes, U., and Essrig, I. M. Some reflections on surgical principles in treating cancer of colon and rectum: based on analysis of 70 patients. *Ann. Surg.* **130**:1008-1023, 1949.
27. Aylett, S. O. Value of sphincter-preserving operations in cancer of rectum: with special reference to reconstructive abdomino-perineal incision. *Brit. M. J.* **2**:728-731, 1949.
28. Best, R. R., and Blair, J. B. Sphincter preserving operations for rectal carcinoma as related to anatomy of lymphatics. *Ann. Surg.* **130**: 538-556, 1949.
29. Papers of American Proctologic Society and Section of Proctology of the Royal Society of Medicine of England. *Am. J. Surg.* **79**: January, 1950.

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

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CASE 36111

PRESENTATION OF CASE

An eighty-year-old woman was admitted to the hospital complaining of a right-lower-quadrant ache.

Two years before admission, she noted the presence of a mass in the lower abdomen on the right. It was slightly tender to palpation and did not change in size over the period of her observation. During the two years before admission she also noted lower-back pain (in the region of the lumbosacral ligaments), which persisted and, in the few months before admission, confined her to bed. In addition, she had had some vague abdominal "soreness" unrelated to meals or position. Appetite and strength decreased during the year before admission. Because of the loss of her teeth she had not eaten meat for several years and had subsisted on soft foods and soups. Four days before admission the right-lower-quadrant mass became painful. A physician was called, and hospitalization was advised.

The bowel history was indefinite, but she had always been constipated, and this had increased following prolonged bed rest. There were no other gastrointestinal or other symptoms.

Physical examination showed an asthenic, poorly nourished woman in no acute distress. The skin was dry and wrinkled. Arcus senilis was present bilaterally, as were beginning cataracts. She was edentulous. The anteroposterior diameter of the chest was enlarged, and there was a dorsal kyphosis. The heart was not enlarged, and a Grade II precordial systolic murmur was present. In the abdomen a doughy, large, freely movable, slightly tender, firm, right-lower-quadrant mass was felt. Its borders were definite laterally, superiorly and inferiorly. Its medial border was indistinctly made out just across the midline. The surface was firm, with many irregularities, especially inferiorly, where the mass rested on the iliac crest. On the superomedial aspect of the mass was a questionable, additional, irregular, separate mass, which was rubbery and firm. Pelvic and rectal examinations were not remarkable except for the intra-abdominal mass, which could be palpated from the right vault.

The blood pressure was 165 systolic, 75 diastolic.

The hemoglobin was 14 gm. The white-cell count was 10,500, with 74 per cent neutrophils, 25 per cent lymphocytes and 1 per cent monocytes. The urine was normal. A stool was guaiac positive. The nonprotein nitrogen was 35 mg., and the serum protein 5.7 gm. per 100 cc. The chloride was 100 milliequiv. per liter. The prothrombin time was normal. A proctoscopic examination demonstrated no abnormalities. A chest x-ray film showed bilateral emphysema and fibrosis, with a marked increase in the anteroposterior diameter of the chest. The heart was not enlarged. There was a marked sclerosis and tortuosity of the aorta. A lateral film of the thoracic spine showed marked osteoporosis and a slight wedging of the bodies of the sixth and eleventh thoracic vertebras. There was no definite evidence of bone destruction. A lateral film of the skull showed no evidence of metastasis. In a plain film of the abdomen a lobulated, soft-tissue mass was present in the right lower quadrant. No fecal material was seen within it. It appeared to be outside the large bowel and displaced the cecum upward and toward the midline. There were three irregular calcifications overlying the midabdomen. All the bones showed osteoporosis, and there was a rotary scoliosis of the lumbar spine, with hypertrophic changes at the joint margins. Intravenous pyelograms demonstrated that the kidneys were within normal limits in size, shape and position. The mass in the right lower quadrant was separate from the right kidney. It appeared to have displaced the right kidney upward and the right ureter toward the midline. There was a slight dilatation of the calyxes and pelvis on the right side. A barium enema was attempted but was unsatisfactory. A single film showed the barium to have entered as far as the splenic flexure without delay.

On the eleventh hospital day an operation was performed.

DIFFERENTIAL DIAGNOSIS

DR. JOHN SPROULL*: I do not know what a man of my experience means by allowing himself to be induced by Dr. Castleman to come down here. I am the oldest man and the oldest practitioner of medicine in this room. I have been practicing for forty-eight years. I should have known better because this is the land of mistakes.

The assumption that this was a tumor that was getting tender is of some importance. If this patient were a male we would be entitled to believe that a tumor arose from tissues in its immediate vicinity.

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The converse is true of females, who notoriously have abdominal tumors that arise in the true pelvis and may be found in any part of the abdomen — in the left hypochondrium, the right hypochondrium and the epigastrium. Such tumors in females are known to migrate throughout all parts of the abdomen; therefore, the sex of the patient is important.

I would like to ask if the patient was a native-born American.

DR. TRACY B. MALLORY: She was born in Ireland.

DR. SPROULL: That is pretty near. If she was born in Scotland it would have been better.

The fact that she noted the mass for two years before admission does not mean that it was only of two years' duration; it may have been present for as long a period as thirty years. I submit the reason that the patient felt the mass was not because of any sudden growth of the tumor but rather because she had grown thin with age and the abdominal wall had shrunk so that the tumor became more apparent. The fact that she did not observe any change in the size of the mass does not mean much. Self-examination of the abdomen is a very difficult procedure and is of questionable value.

It is stated in the record that this patient was edentulous and had to live on soft foods. This does not necessarily mean that her diet was inadequate. Any patient may have an adequate diet with soft foods: the carbohydrate component of the diet may be obtained from cooked cereals, the protein element from the eating of eggs and egg dishes, and the fat elements by the eating of foods prepared with butter or cream. Dr. Richard Cabot, who initiated these clinics, once made the statement that the most common cause of loss of flesh in elderly people was generalized arteriosclerosis. We make a great deal of fuss about arteriosclerosis in the lower extremities, in the brain and in the coronary arteries. Most elderly people have arteriosclerosis of the intra-abdominal blood vessels, which results in an inadequate blood supply to the gastrointestinal tract, and I suggest that more attention should be paid to this condition as a cause of many of the digestive complaints that elderly people suffer from. It is not necessary for such patients to have cancer or any other serious organic condition to explain the digestive disturbances that afflict them.

In the record, the tumor under consideration has been described as being doughy and firm. I submit that a doughy mass is not firm. A doughy mass is one that can be pressed upon with the fingers and whose residual indentations can be felt. The two adjectives are not consistent. I am not trying to be facetious or fault finding. It is very difficult by palpation to tell whether a mass is solid, or firm or cystic.

The "questionable, additional, irregular, separate mass" mentioned in the record gave me a great deal of trouble. If the observation is correct, it means that there must have been two tumors, or a daughter tumor in association with the larger tumor. This would widen the field of investigation greatly. It is also stated that the mass at times extended low in the pelvis, but not whether it was ballotable. The record also states that in the plain film of the abdomen a lobulated soft-tissue tumor was present in the right lower quadrant. This is an unusual finding. The word "lobulated" is very important because it led me astray for several days. The term lobulated means divided into lobes by septums. The best example of a lobulated organ is the lung, and yet the outer surface of the lung is not irregular as the outer surface of this tumor was. There are very few, if any, tumors that are completely lobulated. What is meant by the use of the term "lobulated" in the record is nodular. When I replaced the word "lobulated" with the term "nodular," a good deal of light was cast on the problem of the identity of the tumor.

I know how difficult it is to describe what is seen on the x-ray film, and no doubt, I, myself, have rendered many faulty descriptions. I am sure the X-Ray Department did not seriously consider the mass as being in the colon, although a film was submitted to me that showed an incomplete examination of the colon by barium enema. On the film submitted there was no evidence of intestinal obstruction. If a mass of this size was in the right half of the colon there should have been dilated loops of ileum indicating that the mass involved the ileocecal valve and thus prevented the emptying of the contents of the ileum into the colon. I will state categorically that the tumor under consideration was not within, nor did it originate within, the lumen of the bowel. It is important to fix accurately the location of the tumor whether intraperitoneal or retroperitoneal. Large retroperitoneal tumors are relatively uncommon. I have encountered only a very few, such as large lipomas and also large tumors of the retroperitoneal lymph nodes. The tumor, if retroperitoneal, could have been an extension of a dumbbell tumor that had its origin within the spinal canal. None of the retroperitoneal tumors that I have mentioned have shown the marked mobility that this tumor is alleged to have possessed. I therefore place the tumor in the peritoneal cavity. It could have been an ovarian cyst. The fact that pelvic examination was essentially negative does not rule it out. I am not able to rule it out completely, although I do not believe this was an abdominal tumor with its origin in the pelvis. Some ovarian tumors have long pedicles, and some do not have pedicles at all. Dermoid cysts and the intraligamentous cysts do not have pedicles. The large, multilocular, serous cysts very often have pedicles of varying lengths. I do not believe that this was a pedunculated ovarian cyst. The tumor was situated high up in the right side of the abdomen. There is no evidence that it

possessed any lateral mobility and that it could be moved from the right side of the abdomen into the left as pedunculated ovarian cysts often can.

Several other conditions require consideration in the differential diagnosis: firstly, a large gall bladder. In the course of the practice of medicine I have met very large gall bladders. The size of the tumor does not exclude a large gall bladder. I can well remember one gall bladder that had a capacity of twenty-four ounces and contained over 5000 gallstones, but it was not irregular in outline as this tumor was. Nevertheless, a large gall bladder cannot be lightly dismissed.

Hydatid cysts in the liver next came into consideration. I scanned the x-ray films trying to find calcification of the walls such as occasionally is seen in echinococcus cysts, but I could find no evidence of it. Moreover, I had never known of pedunculated echinococcus cysts. I consulted with Dr. Kranes regarding this condition and asked him if he would consult all the books in the Boston Medical Library to find out whether echinococcus cysts ever developed a peritoneal pedicle. He was not able to find any statement whether they did or did not. My own library was not adequate for such research. My experience with echinococcus cysts is limited. I have seen some in my travels and journeys to various clinics. All echinococcus cysts that I have seen were either firmly attached to the undersurface of the liver or imbedded in the substance of the liver. The lack of calcification in the tumor does not positively exclude an echinococcus cyst. I believed that I could, with reason, discard this diagnosis.

The next condition for consideration was pancreatic cyst. I have observed a number of pancreatic cysts in my own practice and have seen a number of them in other clinics. I have not observed a pancreatic cyst located strictly in the right side of the abdomen. Some do start just above the umbilicus, some just below it, and some just behind it. I know of no pancreatic cysts that arise solely from the head of the pancreas. Do you, Dr. Mallory?

DR. MALLORY: I cannot remember one.

DR. SPROULL: I therefore discard the diagnosis of pancreatic cyst, although the alleged mobility of this tumor does not exclude that possibility. The pancreas can be moved in a cephalocaudal direction so that a cyst or tumor arising from the pancreas may possess a reasonable degree of mobility in the cephalocaudal direction, but pancreatic cysts do not, as a rule, have much, if any, lateral mobility. It is very important to remember that the pancreas is a movable organ. I learned that only fifteen years ago.

Another condition worthy of mention in the identification of this tumor is mesenteric cyst. By and large, in small communities like the one I work in, mesenteric cyst would be regarded as a very rare disease. I have never encountered one in my own practice, but have seen one or two in some of the large clinics that I have visited. Mesenteric cysts are probably the most movable of all intraperitoneal tumors. They can be irregular in outline. However, I doubt that this was a dermoid cyst of the mesentery, despite the calcified shadow noted in the right iliac fossa, which resembles a tooth. The X-Ray Department failed to include this tooth-like shadow in its report. This failure may be a conspiracy of silence on the part of the X-Ray Department. Having discarded all the various diagnoses that I have discussed, I believed this tumor, which was said to have possessed marked mobility, must have had an attachment either at its proximal end or at its distal end. If the attachment was at the distal end, it must have been a pedunculated ovarian cyst, and that diagnosis I have already discarded. Therefore, I believed that the most likely organ in the peritoneal cavity that this tumor could have been attached to was the undersurface of the liver. I could not rule out positively echinococcus cyst. If the patient had been a native-born American that would have helped some. I believed, however, that this was some type of tumor that arose from the undersurface of the liver.

I spoke about the large gall bladders that I had met, not too uncommonly, and certainly, with any tumor in the right side of the abdomen, the gall bladder must be given consideration. One of the characteristics of the tumor that bothered me was the fact that it was irregular in outline, not lobulated, but nodular. It took me a long time to get over that. I looked at the x-ray films, to see if I could find gallstones. Were there gallstones present or not? If one looks long enough at x-ray films one can see almost anything, even an angel. While I was studying the tumor, it occurred to me that I was making a great deal out of a case that was probably relatively simple. I had always taught that one should expect the usual and be familiar with the usual and know where to go to find information about the unusual. I believed, therefore, that this tumor was probably something that had come within the range of my own experience. I was sure that it was an intraperitoneal tumor and that it originated from the undersurface of the liver. Included among the x-ray films that were sent to me for study was an excellent one of the kidneys both by intravenous and by retrograde pyelography. These examinations clearly demonstrated that the tumor was not connected with the kidney. It evidently had invaded the right iliac fossa, the right flank and also the right paravertebral gutter. I believe the extension into the right paravertebral gutter could have accounted for the displacement of the kidney by an intraperitoneal tumor and made it possible for the examiner to feel the medial margin of the tumor. The tumor had no lateral mobility. To account for the displacement medially

of the kidney and of the ureter by an intraperitoneal tumor it is necessary to take into consideration the physical state of this patient. She was an elderly woman who had lost all her subcutaneous fat—preperitoneal fat, retroperitioneal fat and the fat that surrounds the kidney and holds that organ in place. The fact of the matter is that this tumor had no mobility of itself. The mobility that it exhibited was the mobility of the organ to which the tumor was attached.

Among other films sent for my consideration were posteroanterior and lateral views of the chest. The chest film showed the presence of marked pulmonary emphysema. In addition, the lateral view of the chest showed marked decalcification of the bodies of the dorsal spine. The roentgenologic findings of pulmonary fibrosis and pulmonary emphysema shown on the roentgenologic examination of the chest are no aid whatever in helping to identify the tumor in the abdomen. However, they may be of importance as factors in understanding the limitation of this patient's activities. The decalcification of the dorsal spine noted has no relation whatever to the diagnosis, but it is of some importance to call it to attention as a very common condition about which we know very little. It happens that I am interested in the diagnosis and recognition of this condition, and I believe that the time has come when concerted efforts should be made to establish standards of what constitutes decalcification, and also the causes of such decalcification, and whether any therapeutic measures can ever produce recalcification of the decalcified bones of the spine. Such decalcification of the dorsal spine is a frequent cause of disabling and painful backaches in females. It is seldom discovered before it is very far advanced and very disabling. Another condition is clearly visualized on the roentgenologic film, and this was not mentioned in the x-ray reports. This patient had degenerative disease of the right hip joint. This disease is very painful, and patients suffering with it have marked limitation of their motor activities as a result of its presence. There is also seen on the x-ray film a calcified shadow, which I believe is a calcified mesenteric lymph node. If this is correct, the shadow has no clinical significance. Reverting to the discussion of the importance of pulmonary emphysema, pulmonary fibrosis and the painful condition of the right hip joint, I submit that a patient eighty years of age with severe emphysema and painful involvement of a hip joint can decide to stay in bed, not because of the tumor but because of the disability these two conditions would mean. I am sure that this patient could not move from bed to chair without being short of breath, or from chair to bed without severe pain in the involved hip. There is no evidence that the tumor was a cause of discomfort, or that the tumor limited the motor activities of the patient. I believe that I have now discussed most of the important factors that should be considered in the differential diagnosis of this tumor. It is best not to extend oneself too much. One seldom, if ever, gets into trouble by maintaining silence.

After considering all the various conditions that have already been described, I arrived at the opinion that this tumor had its origin on the undersurface of the liver. I cannot rule out, with certainty, an echinococcus cyst. I cannot say that it is not a gall-bladder tumor, although it does not correlate with that of any gall bladder I have ever encountered. A neoplastic gall bladder, in my experience, is not nodular. The carcinomatous gall bladders I have seen have had no evidence of neoplasm on their peritoneal surfaces. The cancer only became evident when the interior of the gall bladder was inspected after incision of the walls. The evidence in the cancerous gall bladders that I have seen consisted of erosion and ulceration of the interior surface and cauliflower growths of varying size filling the lumen. It is possible that this tumor represented a cancerous gall bladder or some unusual tumor-like sarcoma of the liver. I have met with 2 cases of sarcoma of the liver that were large, irregular tumors on the undersurface of the liver. Such sarcomas can attain great size, and their diagnosis is practically impossible except by exploratory laparotomy. This tumor does not correspond to my idea of what constitutęs hydrops of the gall bladder, nor what is known as a surgical gall bladder. If I could visualize gallstones in some of the films that were presented for my examination it might influence me greatly in my leaning toward a cancerous gall bladder. My final diagnosis, therefore, is a tumor originating on the undersurface of the liver. I am not prepared to state the type. I believe the mobility of the tumor was dependent on the mobility of the liver and that the tumor had no inherent mobility. I would not be surprised if it turned out to be one of the unusual tumors like sarcoma, although the age of the patient is against it. Dr. Elmer Bagnall used to say that there are no "evers" and no "nevers" in the practice of medicine.

DR. WALTER BAUER: Did you say the fact that she was a woman was a help?

DR. SPROULL: It was not any help. On consideration, you will admit that the female sex is notorious for many pedunculated tumors that occur in the abdominal cavity. The same statement is not true of males. Pedunculated tumors occur within the gut in males but not, so far as I know, in the peritoneal cavity.

DR. STANLEY M. WYMAN: Were you intrigued by the shadow of calcification in the midabdomen?

DR. SPROULL: I would not call that the midabdomen. These areas of calcification are in the right lower quadrant. I thought at first that this shadow was the shadow of a tooth.

DR. WYMAN: That thought occurred to me too regarding the lower calcification, but I was more intrigued by the one calcified shadow over the spine.

DR. SPROULL: That did not bother me. I thought it was a calcified mesenteric lymph node such as we often see.

DR. WYMAN: That is a gallstone.

DR. SPROULL: Perhaps it is, — I do not deny the possibility, — but I would say calcified mesenteric lymph node in that area.

CLINICAL DIAGNOSIS

Carcinoma of ovary?

DR. SPROULL'S DIAGNOSIS

Tumor of undersurface of liver.

ANATOMICAL DIAGNOSES

Adenocarcinoma (Grade III) of gall bladder, with invasion of liver.

Cholelithiasis.

PATHOLOGICAL DISCUSSION

DR. MALLORY: Dr. Ingersoll, will you tell us what you found at operation.

DR. F. M. INGERSOLL: The mobility of this tumor, as Dr. Sproull said, was dependent on the fact that it was attached to a very mobile liver. We were fairly sure that it was not a pelvic mass. However, we did not make the diagnosis nearly as well as Dr. Sproull has. We thought it was probably a retroperitoneal tumor, because of the displacement of the ureter and of the kidney. One of the assistant residents thought we should make a retroperitoneal type of incision and not enter the peritoneal cavity. I believed we could best operate, even if this were a retroperitoneal tumor, through the peritoneal cavity and was surprised upon opening the cavity to find that it was an intraperitoneal tumor that was adherent to or part of the liver. It was, as Dr. Sproull has said, a carcinoma of the gall bladder. It was a large tumor, as one can see from the x-ray film. It was nodular rather than lobular.

DR. SPROULL: I have one last word to say. I had great difficulty at first in explaining the displacement of the kidney. By and large, displacement of the kidney and ureter means retroperitoneal tumor, but this patient had lost all her subcutaneous, preperitoneal and retroperitoneal fat, and the fat that holds the kidney in position. I am sure that it would have required only a minimum amount of intraperitoneal pressure to displace the kidney.

DR. MALLORY: The gall bladder was markedly enlarged, and its walls were so nodular and thickened by neoplasm that the organ was scarcely recognizable until it was sectioned and numerous gallstones were found embedded in papillary masses of tumor tissue. The tumor had invaded the liver over a broad area, and it was necessary to resect a considerable wedge of liver substance to remove it. I cannot settle the debate between Dr. Wyman and Dr. Sproull about whether the shadows noted in the x-ray films were due to the gallstones or to calcified mesenteric lymph nodes.

CASE 36112

PRESENTATION OF CASE

A forty-six-year-old housewife was admitted to the hospital because of an abdominal mass.

Two and a half months before entry, during a violent spell of coughing, the patient suddenly experienced a sharp, rather severe pain in the left upper quadrant of the abdomen. Upon palpating that area she discovered a large mass. From that time on she had intermittent attacks of pain in the left upper quadrant and was constantly aware of the presence of the mass, which seemed to vary in size. Two months prior to admission the patient suddenly developed frequency and dysuria, with gross blood noted on the toilet paper on several occasions after voiding. There were no chills or fever. Since that time there had been two other episodes of dysuria, the last occurring two weeks before admission. Between attacks she was completely symptom free except for the left-upper-quadrant mass. She had lost some 14 pounds in weight during the three months before admission. She had been anorexic during the final two weeks, but had vomited only once. There had been no change in her bowel history; movements were regular and without bleeding. The patient thought the mass had decreased in size since it was first noted.

The past history was long and complex, and she had had nine previous admissions within the last three years. During the third year before this entry there were three admissions, two within a six-week period because of acute alcoholism, and the third, for pneumonia of the left lower lobe. A needle aspiration of the liver during the first admission had shown findings consistent with acute progressive alcoholic cirrhosis. During the second year before this entry there were two admissions for acute peripheral neuritis and anxiety state. In the year before this entry she was admitted for a period of three weeks, during the first two weeks of which she ran an irregular fever. She complained of having had fatigue, anorexia, headache and soreness in the neck for three days before entry. The physical findings were those of slight, tender, cervical lymphadenopathy, scattered squeaky, musical rales in the chest and a palpable liver. During one six-day period the hemoglobin fell from 13.0 gm. to 4.5 gm. per 100 cc. She became jaundiced, and the spleen became palpable. The white-cell count ranged from 4700 to 11,500, and the highest percentage of neutrophils at any time was 71. On several occasions the blood smear

showed moderate achromia and considerable variation in size of the red blood cells but no variation in shape; up to 5 per cent atypical lymphocytes, 1 per cent atypical monocytes and 4 per cent blast forms were seen. The heterophil-antibody and Widal tests were negative. Several prothrombin-time studies were normal, and a serum bromsulfalein test showed 14 per cent retention of the dye in 1 hour. The serum van den Bergh was 0.5 mg. per 100 cc. direct and 0.9 mg. indirect, and the cephalin flocculation was ++ in 24 and 48 hours. During the final week of hospitalization the temperature remained normal, and after several transfusions the patient was discharged much improved. It was believed that she had had a viral infection with secondary acute hemolytic anemia. Six months before the present admission she entered the hospital for one day because of an acute anxiety state. Physical examination was essentially negative. The liver and spleen were not palpable. The urine was normal. The hemoglobin was 16.0 gm. per 100 cc. and the white-cell count 9700, with 47 per cent neutrophils and 51 per cent lymphocytes. Five months before the present admission the patient was brought to the Emergency Ward. Several hours before, she had leaped out of a window, 9 feet above the ground. She had not been unconscious and complained of pain on the right side. On physical examination there was a deep laceration of the scalp over the right occipital area and tenderness over much of the right side of the body, most marked in the right upper quadrant. The latter was of such degree that adequate examination of the liver could not be made. The rest of the abdominal examination was negative. Roentgenograms of the skull, chest, spine, pelvis and lower extremities showed only a fracture of the right sixth rib and right ankle. The patient's course was satisfactory, and she was discharged on the seventh day.

On physical examination the patient was well developed and well nourished. The chest was emphysematous. The lungs were clear, with the exception of a few scattered musical rales and rhonchi. There was a rounded, firm, rather hard mass in the left upper quadrant. This measured about 20 cm. in diameter and extended from just beneath the left costal margin to about the anterior superior spine of the ilium and posteriorly to the spine. It was freely movable and nontender, did not pulsate and did not descend with inspiration. On deep palpation one could get the fingers between the mass and the lower rib margin. The right kidney and edge of the liver were both palpable.

The temperature, pulse and respirations were normal. The blood pressure was 130 systolic, 80 diastolic.

The urine was normal. The hemoglobin was 13.4 gm. per 100 cc., and the white-cell count 11,000, with 73 per cent neutrophils. There were moderate anisocytosis and a rare stippled cell. The nonprotein nitrogen was 23 mg. per 100 cc. A stool specimen was guaiac negative. Cystoscopy and a left retrograde pyelogram were negative. An intravenous pyelogram could not be done because of an allergic history. A roentgenogram of the chest was normal except for callus formation about the fractures previously described. A plain film of the abdomen showed the left kidney to be considerably higher in position than previously. There was an indefinite soft-tissue fullness overlying the left abdomen and iliac crest. The lower portion of the left-psoas-muscle shadow was observed. The body of the stomach appeared to be displaced upward. A barium enema showed easy filling of the colon, with no obstruction; the mass on the left side of the abdomen compressed the ascending portion of the colon and displaced it laterally (Fig. 1). There was also upward displacement of the left side of the transverse colon but with no evidence of tumor involving the bowel wall. A gastrointestinal series was negative except for upward and medial displacement of the stomach, duodenum and jejunum by the mass.

On the seventh hospital day an operation was performed.

Differential Diagnosis

Dr. Arthur W. Allen*: Could we see the x-ray films, Dr. Wyman?

Dr. Stanley M. Wyman: The soft-tissue mass can be seen in the left lower abdomen as a poorly defined dense shadow overlying the iliac crest and causing perhaps some displacement of the lower ureter. The left kidney is well seen and lies higher than it did on previous admissions. A subsequent film taken at the same time, but not shown now, revealed normal left urinary passages. One cannot

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see with certainty the outline of the spleen on any of the films that we have at hand nor on any of the others on file in the X-Ray Department. The transverse colon, as described, is displaced upward in its distal portion, and there is thinning and compression of the middle descending colon by the soft-tissue shadow (Fig. 1). The colon, however, shows a normal mucosal pattern, with no evidence of intrinsic involvement.

DR. ALLEN: We have a patient who had been under observation for three years with several hospital admissions so that we must assume that this tumor, which she felt two and a half months prior to her last admission, was of fairly recent origin. She entered the hospital for one day six months before the last admission, and the record states that the liver and spleen were not palpable at that time; had there been a lesion of any size, I think it would have been felt.

The next feature that strikes me as important in the very long history is the accident. She fell a distance of 9 feet from a window to the ground with sufficient force to produce a laceration of her scalp, and fractures of the right sixth rib and right ankle. At that time she had so much tenderness throughout the abdomen that a satisfactory examination probably was not possible inasmuch as the liver edge could not be felt and it had been felt previously.

The next thing that I must explain is the period of dysuria with gross blood noticed after voiding. One would have to be very suspicious of a kidney lesion under these circumstances, but it is difficult to explain this on the basis of a kidney lesion because of the normal retrograde pyelogram. I think that it would be possible to explain that type of bleeding and dysuria as completely unrelated to the kidney but perhaps due to a urethral caruncle or something of that sort, which would account for both the intermittent dysuria and the bright-red blood on the toilet paper. It was not mentioned that blood was found in the urine at any of the previous admissions, and apparently it was not recorded here on the last admission.

The patient was an alcoholic, and a positive diagnosis of cirrhosis of the liver was made approximately three years prior to the last admission. It would be interesting if one could tie this situation to the present tumor. From the patient's standpoint the sudden appearance of a tumor following a severe bout of coughing strongly suggests hemorrhage into something that was already there rather than a ruptured vessel or aneurysm of sufficient size to explain the mass. If the hemorrhage alone had caused a tumor of this size to develop suddenly, I believe it would have created a state of shock. The patient would have had such tenderness over it that she would not have been able to discover the mass herself. I suspect that the episode of noting

FIGURE 1. *Barium-Enema Examination.*
Arrows point to the compression of the mid-descending colon. (Note the upward displacement of the distal transverse colon.)

it after a severe bout of coughing merely means that she had not felt her abdomen — or no one else had — after the tumor developed and that probably an adhesion did break loose or possibly a small hemorrhage within the tumor occurred. It is hard for me to believe that a tumor of this size could have developed in such a short period that the patient would have noticed it immediately after feeling discomfort following a severe bout of coughing.

The various types of tumors in this region are many. I will speak of the least likely things first. An aneurysm must always be considered, but this

tumor definitely did not pulsate, and I think if it had been an aneurysm there certainly would have been transmitted pulsations. A cyst of the kidney seems unlikely because I doubt if the pyelogram would have been normal even with a cyst of the lower pole.

One must always consider lymphoma and lipoma in the retroperitoneal area when any solid tumor is palpated in the abdomen. This almost certainly was not retroperitoneal because the record states very clearly that it was freely movable. Lymphoma often produces a tumor that is discovered by the patient or on physical examination by the physician, but it is nearly always tender to pressure and it can be quite movable. The other day we saw a patient whose entire stomach and transverse colon had been removed six years ago in this hospital for what we considered very unfavorable carcinoma that turned out to be lymphoma. This mass was discovered on routine examination by Dr. Waddell, who at that time was in his first year of internship here. The patient had come to the hospital for an orthopedic condition.

Recently we have seen a woman who had a uterine fibroid that filled the entire right abdomen all the way from the liver edge down to the crest of the ilium, and the pelvis so far as we could tell was completely clear except for two or three small fibroids. She had had two cesarean sections, and the uterus was fixed to the abdominal wall and the fibroid had grown upward in this manner. I might say that none of us made that diagnosis. I doubt very much if this was a uterine fibroid on the left side of the abdomen, although fibroids can create the same sort of picture as that described in this case.

We have also seen, in the last year or two, a large chylous cyst that was noticed on routine physical examination. This was placed almost exactly where this tumor that we are talking about was. It displaced the organs very similarly and was freely movable, nontender and solid. It arose from the upper jejunum. A mesenteric cyst of the ordinary type, however, is very much more common. Of course both a chylous cyst and a mesenteric cyst could occur in a period of two or three months although I would have supposed that either of these might take a little longer. Pancreatic cysts occur following injury to the pancreas so that it seems likely that this woman, who had an accident, could have developed a large pseudocyst of the pancreas. It would fit perfectly into this displacement of her adjacent structures, but it extended farther down into the abdomen than any pancreatic cyst that I have seen and also it was too freely movable for a pancreatic cyst. Pancreatic cyst might conceivably fit the picture better than anything else I can think of on the basis of its having decreased and increased in size, if we can credit this patient's story.

Then we come down to something arising from the left lobe of the liver or from the spleen. A tumor of this size of the left lobe of the liver would be very unusual. Could it have been a tumor of the spleen? The protocol states very definitely that the examiner could get his hand between the border of the left costal margin and the top of the mass, which with splenic masses of this size has been uncommon. However, I believe that a tumor called a hamartoma has been found in the spleen very much like this, and we certainly have seen it in the left lobe of the liver. One would expect that a freely movable tumor of the spleen, instead of being in front of the colon, might have pushed the splenic flexure downward as is usual in splenomegaly, although if the spleen was normal and a tumor arose from its lower pole, it could produce this pressure defect.

I will conclude by making a diagnosis of a tumor of the lower pole of the spleen.

Dr. Fletcher H. Colby: Could I ask Dr. Wyman if he can tell from the x-ray appearance of this mass whether it was in front of the sigmoid?

Dr. Wyman: I cannot tell from these films alone, but I was told by the fluoroscopist that it did lie in front of the descending colon and that the descending colon was pushed back.

Dr. Daniel S. Ellis: I think that was right.

Dr. Wyman: We thought of a mesenteric cyst and secondly of a possible ovarian tumor, very high, but mesenteric cyst was our best bet. We did not think it could be the spleen.

Dr. Wyman: For the same reason that Dr. Allen gave — namely, that the splenic flexure is usually pushed down by an enlarged spleen.

Dr. Ellis: This patient walked into my office and said she had been afraid of coming in two months previously because we had seen her so much in the past two or three years that she thought we would say she was hysterical. When she came in,

she said she had a mass in her abdomen, which she thought was a tumor of the spleen and had better have out. She gave a very definite history of dysuria associated with the pain in the left upper quadrant. On palpation of the abdomen the mass was the size of a grapefruit, and one could get between it and the lower costal margin. It was also easily palpable bimanually. Before she entered the hospital I wondered if we were dealing with a tumor involving the left kidney. After cystoscopy this seemed to be ruled out. Our reasoning was much the same as Dr. Allen's. We operated with the preoperative diagnosis of mesenteric cyst or pancreatic cyst, although the latter was unlikely because the mass was so freely movable. Just before she went under ether, I wondered if it were an ovarian tumor because it seemed much lower than before.

Clinical Diagnosis

Mesenteric cyst.

Dr. Allen's Diagnosis

Tumor of lower pole of spleen.

Anatomical Diagnosis

Splenic cyst, traumatic.

Pathological Discussion

Dr. Tracy B. Mallory: This patient was operated upon by Dr. Robert R. Linton, who found that the tumor was in fact a very much enlarged spleen. I have a picture of it here (Fig. 2). Two thirds of the spleen consisted of a rather thin-walled cyst, which was in the lower pole. The cyst was filled for the most part with gelatinous exudate, but at one point a focus of relatively fresh blood clot was present, which I think represented bleeding into the cyst in some fairly recent period. The cyst had no lining cells, the walls consisting only of fibrous tissue with moderate hemosiderin pigmentation. This is a so-called false cyst of the spleen and is usually associated with a history of trauma. It probably forms from a hematoma by much the same mechanism as a subdural hematoma, which progressively grows in size as protein molecules in the clot disintegrate and the osmotic pressure of the material increases and attracts more fluid.

Figure 2.

Dr. Allen: Then the patient was right in her statement that it did change in size with each bout of hemorrhage?

Dr. Mallory: It is at least possible.

Dr. Gordon A. Donaldson: Would you explain the hematuria?

Dr. Ellis: She did not have hematuria after admission — the urine was normal in the hospital.

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EDUCATION AT HARVARD

THE channels of education are perpetually changing with the changing needs of the times and the increasing volume of knowledge that must flow through them. Reform is indeed a constant, if education is not to become static.

These points are brought out in the annual report of President Conant of Harvard, released in January, 1950. The revolutionary event in the administration of his predecessor, President Lowell, was the acceptance of the premise that a college education must involve both concentration and distribution. This principle was a departure from the free elective system introduced by President Eliot, without implying a return to the rigidly prescribed curriculum of the early nineteenth century.

The system of concentration and distribution has also been subjected to constant scrutiny and finally an official review by a committee on general education. The college, as a result, has committed itself to a new program that recognizes the dangers of specialization by requiring each undergraduate to include in his program three courses in general education. These are to include the humanities, the social services and the natural sciences.

Each student prior to graduation, and preferably in the early years of his training, must thus have exposed himself to the influence of academic culture in its broadest sense; he must have taken at least three prolonged flights that will reveal the topography of the terrain in which the accumulated knowledge of the world resides.

Exceptions are to be made in the case of those who plan to study medicine; whether these exceptions represent rewards or penalties is yet to be determined, for no one more than the physician requires breadth in his education if medicine is to survive as a learned profession. Moreover, as was brought out in the meeting of the academic deans earlier in the year, the intention of an undergraduate to study medicine may have little bearing on his fitness or his chances for acceptance for that arduous course of study, to which so many believe themselves called, and for which so few are actually chosen.

The cost of education is of primary concern to all universities today, affecting the wealthiest as well as the poorest. This high cost applies most particularly to the study of medicine and the allied sciences, and, in the medical area, teaching and research in public health are suffering most severely. That this need must be met is obvious, because of the obvious fact that public health and preventive medicine represent the currents in which the medical practice of the future will flow.

The relations of a medical school to its affiliated teaching hospitals, themselves in straitened circumstances, need constant attention; these are among the problems raised in part by the prodigious strides that have been taken in the medical sciences.

The cost of the benefactions that science has brought to mankind must not be allowed to become prohibitive, nor must medical education become a captive branch of learning through the unguarded acceptance of those doles that eventually lead the way to universal "welfare."

FIFTY YEARS OF MEDICINE

THE *British Medical Journal* has devoted a large part of its first issue of 1950 to a review of the advances in medicine made during the last half century. And what a period of progress this has been, along the united front of medical science in the service of mankind!

The opening paper, "Advances in Medicinal Therapeutics," contributed by Sir Henry H. Dale, outlines the progress that has been made in immunotherapy, chemotherapy and deficiency replacement. Geoffrey Jefferson, professor of neurosurgery at the University of Manchester, after a brief description of the surgery of fifty years ago, comments on the contributions for which the Great Wars were responsible, then lists the accomplishments of the Age of Specialism. Although this type of progress has been altogether desirable, as well as inevitable, it is perhaps to be regretted that there is left "too little opportunity for the general cultural self-education so necessary to happy existence and one that should continue throughout life."

Obstetrics, as related in "Fifty Years of Midwifery," by R. W. Johnstone, has advanced as fast and as far as any branch of medicine, for it has risen to the rank of a science from the almost routine matter of attending on a physiologic function. This change has come about largely through the acceptance by society of a new point of view, what was a private female performance with one gentleman, perhaps, as observer, assistant or meddler, has become in part the responsibility of the physician. A quotation from Playfair illustrates the attitude from which the departure has been made: "It is often sufficiently easy to recognize a pelvic presentation by abdominal examination *if we have occasion to make one.*"

The impressive inventory of progress reports concludes with one on "Medical Progress from 1850 to 1900" by the eminent writer on medical history, Charles Singer, professor emeritus in the University of London. In considering the fruitful second half of the nineteenth century, Singer points out that "History does not really accommodate itself to those exact dates and places that we learned at school." An event, which man likes to link to a specific date, is subject to the rules of relativity; time is consumed in its preparation, and its effects are without end.

The *British Medical Journal* thus has flashed back upon the screen those advances that first came into view in the opening half of the twentieth century, which, like that period of time (for a few more months at least), have not yet come to their conclusion.

HELP FOR THE ALCOHOLIC PATIENT

OFFICIAL recognition of the now commonly accepted thesis that alcoholism is a medical and public-health problem and not a purely correctional matter is found in the annual message of Governor Paul A. Dever to the two branches of the Legislature on January 4, 1950. In this message the Governor states that, "Chronic alcoholics often receive in the course of a lifetime as many as 200 repeated jail sentences at great expense to the state and little, if any, benefit to the individual."

Governor Dever refers to the report of the Commission on Alcoholism, which states that there are in Massachusetts 30,000 alcoholic patients and at least 90,000 problem drinkers many of whom are potential alcoholic patients. Referring to the important constructive work in other states in alleviating and solving the problems for many persons through clinical programs, His Excellency finds encouragement in the offer of several of the general hospitals to establish such clinics under the direction of the State Commission on Alcoholism provided that the State bear the cost of personnel furnished.

The Governor further states that, "If the 50 to 75 per cent success which such programs have enjoyed with the patients seen up to now is only half realized the savings to the state and the salvage of those who would otherwise suffer ruination will more than repay for the modest expenditures required to operate such clinics."

His Excellency is to be congratulated when, in conclusion, he proposes that the Legislature establish such clinics and promises to include in his budgetary recommendations for the Department of Public Health an item to cover their cost.

MESSENGERS OF GOODWILL

THE Medical and Surgical Relief Committee, Incorporated, of America was first organized in 1940 under the stress of war conditions elsewhere in the world, and was incorporated in 1947 with Fleet Admiral William F. Halsey as president.

Its purpose has been and is "to meet authentic appeals from any recognized agency, hospital, welfare group or mission, in any part of the world, regardless of race, creed or color." In exchange for this aid the beneficiary must guarantee only to give medical or dental service free to the needy.

During the first eight years of its activities, or until January 1, 1949, a total of well over a million dollars in medical and surgical supplies have been shipped through the New York office to areas that are in desperate need in every part of the world. In addition to money, all types of drugs, dietary supplements, hospital ware, recent medical and dental publications and every sort of new or cast-off but usable instrument from hypodermic needles to microscopes are in urgent demand.

Locally, the Woman's Auxiliary to the Suffolk District Medical Society has already taken over the collection of such supplies from doctors' offices, and plans are on foot to extend the organization throughout the Commonwealth. Physicians are urged (and are urged to urge their secretaries) not to throw away drug samples or discarded instruments but to put them in the way of going their "second mile" of usefulness to mankind.

Andrew H. Brand, who died of pneumonia, recently, in Kentucky, aged 16, weighed 500 lbs!
Boston M. & S. J., *March 13, 1850*

MASSACHUSETTS MEDICAL SOCIETY

DEATH

SCHWARTZ — Maurice R. Schwartz, M.D., of Fall River, died on February 20. He was in his sixty-eighth year.

Dr. Schwartz received his degree from Jefferson Medical College of Philadelphia in 1907. He was formerly city physician and assistant physician at the Tuberculosis Hospital.

His widow, two sons and three grandchildren survive.

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SUFFOLK

ADELSON, LESTER, 1163 Boylston Street, Boston.
Tufts College Medical School, 1939.

BULLOWA, MARGARET, 118 Mt. Vernon Street, Boston.
New York University College of Medicine, 1942.

BUSH, RICHARD DAVIS, 524 South 46th Street, Philadelphia, Pennsylvania.
Johns Hopkins University School of Medicine, 1943.

CLARK, DEAN ALEXANDER, Massachusetts General Hospital, Boston.
Johns Hopkins University School of Medicine, 1932.

CREHAN, JOSEPH PAUL, 4 Linskey, Barry Court, South Boston.
Boston University School of Medicine, 1943.

CROCKER, AUGUSTUS THORNDIKE, 37 Chestnut Street, Boston.
Tufts College Medical School, 1944.

FERRINO, PETER J., 77 Bickford Avenue, Revere.
College of Physicians and Surgeons, Kansas City University, 1938. Sponsor: G. Lynde Gately, 624 Bennington Street, East Boston.

GILCHRIST, JOHN JOSEPH, 36 Logan Way, South Boston.
Tufts College Medical School, 1942.

LOVEZZOLA, MICHAEL MARTIN, 73 Barnes Avenue, East Boston.
Tufts College Medical School, 1947.

McDERMOTT, WILLIAM VINCENT, JR., 83 Rogers Road, Hamden, Connecticut.
Harvard Medical School, 1942.

MIKAL, STANLEY, 63 Thomas Park, South Boston.
Tufts College Medical School, 1944.

RAKER, JOHN WILLIAM, 61 Myrtle Street, Boston.
Harvard Medical School, 1941.

SCHWARTZ, WILLIAM BENJAMIN, JR., 27 Longwood Avenue, Brookline.
Duke University School of Medicine, 1945.

SPILLER, ARTHUR ELIOT, 293 Commonwealth Avenue, Boston.
Tufts College Medical School, 1938.

WATSON, CHARLES WESLEY, 51 Phillips Street, Boston.
Yale University School of Medicine, 1943.

WINERIP, BERNARD BENJAMIN, 474 Revere Street, Revere.
Middlesex University School of Medicine, 1937. Sponsor: Harry Silverman, 137 Shirley Avenue, Revere.

Charles G. Shedd, *Secretary*
1180 Beacon Street, Brookline

WORCESTER

ALLAIN, DONALD, Worcester City Hospital, Worcester.
Boston University School of Medicine, 1946.

ASPEN, RICHARD WALTON ALLYN, South Street, Barre.
Tufts College Medical School, 1948.

BAUM, MARVIN, 225 May Street, Worcester.
New York Medical College, 1945.

BEACH, CARROLL CHARLES, JR., 24 Drury Lane, Worcester.
New York Medical College, 1939.

DUNN, JOHN FRANCIS, JR., St. Vincent's Hospital, Worcester.
Tufts College Medical School, 1945.

GRAF, HELEN JULIA, 205 Main Street, Southbridge.
Tufts College Medical School, 1946.

HOLT, EARL KENDALL, JR., Westboro State Hospital, Westboro.
Harvard Medical School, 1944.

KUENSTER, EARL JAMES, 8 Ashland Street, Worcester.
University of California School of Medicine, 1947.

MARSH, CARL GEORGE, 3 Ball Street, Worcester.
College of Physicians and Surgeons, Kansas City University, 1942. Sponsor: Everett P. Jewett, 390 Main Street, Worcester.

MONTGOMERY, DONALD ELON ANGUS, Westboro State Hospital, Westboro.
Tufts College Medical School, 1945.

OUELLETTE, PHILIPPE W., 28 Miles Street, Millbury.
Loyola University School of Medicine, 1942.

REIMAN, EARL ALLEN, 162 Highland Street, Worcester.
Boston University School of Medicine, 1946.

ROY, LOUIS EUGENE, 147 Everett Street, Southbridge.
Tufts College Medical School, 1946.

RUBIN, HAROLD SYDNEY, 19A Elm Street, Worcester.
Tufts College Medical School, 1945.

SEIDENBERG, DANIEL, 9 Sun Valley Drive, Worcester.
College of Physicians and Surgeons, Kansas City University, 1929. Sponsor: Lester M. Felton, 36 Pleasant Street, Worcester.

SLESINSKI, FRANK ALPHONSE, 27 Brownell Street, Worcester.
Southwestern Medical School of the University of Texas, 1947.

SUCHECKI, MARY JOSEPHINE, 1125 Main Street, Worcester.
Boston University School of Medicine, 1945.

VAN GORDON, DONALD J., Worcester State Hospital, Worcester.
University of Minnesota Medical School, 1944.

WOLFSON, IRVING NORMAN, Veterans Administration Hospital, Rutland.
Yale University School of Medicine, 1942.

Donald Hight, *Secretary*
57 Cedar Street, Worcester

WORCESTER NORTH

DONOVAN, THOMAS MAGNER, 42 Fox Street, Fitchburg.
Tufts College Medical School, 1946.

MEYER, FRED MORTON, East Gardner.
University of Halle, 1920. Sponsor: Charles E. Thompson, 80 Glazier Street, Gardner.

PARKER, NORMAN HASTINGS, 22 Walnut Street, Winchendon.
Chicago College of Medicine and Surgery, 1946. Sponsor: Alton B. Skelton, 89 Central Street, Winchendon.

James G. Simmons, *Secretary*
30 Myrtle Avenue, Fitchburg

CORRESPONDENCE

IN DEFENSE OF NATIONAL HEALTH SERVICE

To the Editor: We read that the American medical profession and indeed Americans generally are interested in the National Health Service of Great Britain. The fight of the American physicians against "socialized medicine" is likewise a matter of constant comment in professional circles here. I have followed these matters in your columns and elsewhere for some time — though trained in Canada, I have practiced there and have also worked under the N. H. S. in this country. May I take the liberty of trying to express a point of view that reflects this dual background of experience? I am not an apologist for the N. H. S., and politically I belong to neither the extreme Right nor the Left. I have no personal interest whatever in American developments, and I must ask you to excuse a perhaps too-critical note in my remarks.

Space forbids a lengthy discussion of the N. H. S. I would only say that it is in its infancy, and is undergoing the trials and tribulations peculiar to that period of life. Abuses have occurred, and treatment standards, especially in the matter

of delays in hospital admission, leave much to be desired. Many, if not most, of the dissatisfactions with the N.H.S. on the part of the public are directly attributable to a shortage of skilled staff, especially nurses, and to a general shortage of hospital accommodation, which is the result, in large measure, of the war and its aftermath. From the point of view of the profession, the dissatisfactions have chiefly arisen because of the intransigent attitude of the Ministry of Health in the various points at issue. This is a reflection of the personality of the Minister, and I have little doubt that Mr. Bevan enjoys his present position largely because he was willing and able to deal with the doctors in a manner calculated to nullify the obstructionist attitude of a large section of the profession.

It is, nonetheless, flatly untrue to state that "WHEREAS the experience of all nations where government has assumed control of medical services has shown that there has been . . . a progressive deterioration of medical standards and medical care to the detriment of the people . . ." a resolution that was recently passed by the New Hampshire Medical Society. Presumably, Great Britain was included in this reference, and available statistics fail to bear that contention out.

In the various specialist branches, the very keen competition at each stage of the ascending scale of hospital appointments that eventually lead to full consultant status ensures that the standards are maintained. Indeed, one of the results of the N. H. S. has been a general increase in the salaries paid to junior hospital staffs; financial need has thus been greatly reduced as a deterrent to specialization, and for this reason among others specialist standards are, if anything, rising. True it is that the role of the general practitioner has not yet been stabilized under the N. H. S. There has been a diminution of his responsibility, and the fear has arisen that general practice may become the haven of the disappointed would-be specialist. But is not the American profession, largely as a result of increased specialization, faced with much the same problem?

I have practiced in Canada under a voluntary prepaid scheme providing medical care, and can safely say that abuses were just as frequent as under the N. H. S. This scheme was entirely under the control of the medical profession, and yet needless operations were performed, and superfluous visits made and charged against the scheme. A voluntary scheme administered by the profession does not provide a ready-made answer; the problem of abuses still remains. It is clear that doctors are, if not as other men, at least subject to a degree to their weaknesses.

Can any intelligent view of today's world doubt that the debate between the advocates of complete *laissez faire* and state control is out of date? The problem is how to exercise democratic control; "free enterprise" as understood by John D. Rockefeller has surely been dead since the days of William Jennings Bryan. The truth is that political forces over which the medical profession has no control will demand that financial barriers to adequate medical care be swept away. Citizens will, by and large, tend to vote for the politicians who promise medical care at Government expense; in the current British election, no party has promised to abolish the N. H. S. It does not meet the case to point with pride to the excellent service available to "public" patients; in the final analysis, these are at least in part charitable, and some stigma attaches to their use. In any case, the needs of the medically indigent lower-middle-income group are not met. And no voluntary prepaid scheme will answer, precisely because those who most need care will not voluntarily sacrifice present income to future hypothetical needs. No doubt this is unfortunate, but it is one of the facts with which politicians have to reckon.

In the face of this, the evidence that the American profession is becoming a high-powered political pressure group causes dismay in at least one of your readers. In the process, the spokesmen for medicine seem, at times, to indulge in the same rather meaningless clap-trap as other politicians. It seems senseless to link "socialized medicine" with communism, as has been done in your columns; one might as well attack compulsory state-controlled education as a factor undermining the "American way of life."

British experience indicates that the medical profession is not strong enough to defy a government that, theoretically at least, has the support of the majority of voters. This proposition appears to be self-evident. In its present stand the American profession, or at least that section of it which supports the American Medical Association levy, is gravely prejudicing its future position. Future events may show that these present-day activities are paving the way for an American Bevan, who will be reflexly conditioned to oppose any suggestion, however reasonable, advanced by the leaders of the profession.

Thank you for any attention you may give this letter.

DOUGLAS FINDLAY, M.D.

Leyton, London, E. 10

Note: The proponents of voluntary health insurance and the professionally guided extension of essential medical care to all are not advocates of complete *laissez faire*. It may be hoped that there are few such advocates to be counted in the ranks of the medical profession. Were the opponents on any controversial issue always able to view both sides of the argument dispassionately, there would be less controversy and more discussion and compromise. — ED.

REGARDING BIOSTATISTICS

To the Editor: Dr. James C. McCann points out in his article "Thromboembolism" in the February 9 issue of the *Journal* that upon institution of postoperative Dicumarol therapy there was a decrease in the incidence of thromboembolism as compared with an untreated group.

Upon adding up the two series that he regards as his controls — 15 cases of thromboembolism in 650 patients in addition to 12 cases in 504 patients — one obtains 27 cases of thromboembolism in 1164 patients, an incidence of 2.3 per cent. In his treated series there were 8 cases in 525 patients, an incidence of 1.5 per cent. The author characterizes this decrease in incidence of 0.8 per cent as a "sharp drop."

On statistical analysis, the standard deviation of these series is 0.75 per cent (obtained by the fourfold-table technic), and thus the difference of 0.8 per cent is about one standard deviation from the mean. This difference could consequently occur by chance alone and is *not* statistically significant.

It might be of interest that, at the percentage incidence observed, it would take about 4000 cases in the control and 4000 cases in the treated series to make a difference of 0.8 per cent statistically significant.

HANS W. NEUBERG

College of Physicians and Surgeons, New York, '50

Mr. Neuberg's letter was submitted to Dr. McCann, who offers the following reply:

To the Editor: For me, the rigidities of the statistical method cannot alter the fact that a continuing study of anticoagulant prophylaxis has reduced my rate of embolization in the past two years to 0.4 per cent from the previous rate of 1.3 per cent for the two years noted by Mr. Neuberg and the deaths from massive embolus to 0 for the past two years (prophylaxis), compared with three deaths for the two years noted by Mr Neuberg. If this is "chance alone" I am grateful to chance.

There are qualitative factors in medical practice that counter-balance the straightjacket of quantitative statistical requirements. These are reduced periods of hospital disability and the almost complete disappearance of the distressingly swollen post-phlebitic leg as noted in the office, during postoperative visits. These considerations may move me to submit additional data hopefully, before I have accumulated the suggested 8000 cases — a feat that would drop me right into the middle of my future extracorporeal existence.

JAMES C. MCCANN, M.D.

Worcester, Massachusetts

RESTORATION OF LICENSE

To the Editor: At the meeting of the Board of Registration in Medicine held February 23, it was voted to restore the registration to practice medicine in this Commonwealth to Dr. Samuel C. Zundell, 332 Blue Hills Parkway, Milton.

GEORGE L. SCHADT, M.D., *Secretary*
Board of Registration in Medicine

State House
Boston

BOOK REVIEWS

Children in Conflict: Twelve years of psychoanalytic practice. By Madeleine L. Rambert. With a preface by Jean Piaget. 8°, cloth, 214 pp. New York: International Universities Press, Inc., 1949. $3.25.

In attempting to review this book the reviewer encountered great difficulty owing to a major printing error in the copy at hand in that there was a repetition of printed material between pages 55 and 119 so that several chapters — namely, 2, 3, 4 and 5 of Part II — were to all practical purposes missing. This was rather unfortunate since from the parts available, and especially Part I and Chapter 1, 2 and 3 of Part II, the reviewer was rather encouraged by the clarity in presenting the material for which both author and translator deserve credit. The book is written in a simple style so that it would be easy reading for laymen, physicians and psychiatrists alike, and since controversial material is left out, that which is presented is simple, to the point and concrete. The main theme is that child psychoanalysis can be greatly helped through the use of puppets because children of various age levels are able to express themselves through such a medium and thereby to give a clear understanding of the basis for their conflicts. The examples given are simple and common enough so that anyone can see similarities between the patients discussed and problem children in general.

The latter part of the book available to the reviewer — namely, from Part III on — is extremely vague and from time to time the author seems to express amazement that in her subsequent studies she was able to bear out certain points concerning guilt feelings over sexuality as originally promulgated by Freud. The amazement is difficult to understand.

In brief, this book can serve as a stepping-stone to the families of emotionally involved children and to physicians in general. Unfortunately, the present printing is completely inadequate, and the reviewer strongly hopes that the publishers withdraw from the market existing copies since the book in its present printed form does not do the author justice.

The Origin of Medical Terms. By Henry A. Skinner, M.B., F.R.C.S.(C.). 4°, cloth, 379 pp. Baltimore: Williams and Wilkins Company, 1949. $7.00.

This book should be constantly available to all students of medicine and, be it also said, to every faculty member. Medical students frequently do little else than stitch together scientific words and assume that they are gaining knowledge. The use of terminology in this manner results in freezing the student's thinking. Like the proverbial pie the substance of this book must be carefully digested for its value to be appreciated. It may be said with assurance that students who conscientiously apply themselves to the study of the volume will feel a deep sense of gratitude to Dr. Skinner. The author does not pretend to make this a complete compendium, but the length to which he has carried his effort will bring considerable profit to his readers. One finds an interesting bibliography of the principal sources consulted by the author in the preparation of the book and also a list of the chemical elements with the name and nationality of the discoverer, as well as the date of the discovery. If every practitioner of medicine presented a copy to a student about to enter the study of medicine he would reap a reward for his kindness.

BOOKS RECEIVED

The receipt of the following books is acknowledged, and this listing must be regarded as a sufficient return for the courtesy of the sender. Books that appear to be of particular interest will be reviewed as space permits. Additional information in regard to all listed books will be gladly furnished on request.

Lydia Pinkham Is Her Name. By Jean Burton. 12°, cloth, 279 pp., with illustrations. New York: Farrar, Straus and Company, 1949. $2.75.

This small book tells the life story of a woman who began in a small way in 1873 a medical proprietary business, based on the use of a herb remedy. The business, founded in 1873, has developed into the multi-million Lydia E. Pinkham Medicine Company of today, a concern that spent $800,000 last year for advertising. The details of the inter-family strife for the control of the Company and the controversy with the Federal Food and Drug Administration are given in detail. In 1936 the Company set up a laboratory with research chemists as personnel and has continued active until the present. The story is interesting but rambling and difficult to follow with comprehension. The lack of chapter headings and an index detracts from its value as a reference source. However, the volume should find a place in medical-history collections.

A Year With Osler, 1896-1897: Notes taken at his Clinics in The Johns Hopkins Hospital. By Joseph H. Pratt, M.D., a member of the Class of 1898. 8°, cloth, 209 pp., with 5 plates and frontispiece. Baltimore: The Johns Hopkins Press, 1949. $4.00.

Dr. Pratt, in this volume, presents a reproduction of his notes of clinical cases and lectures of Dr. Osler, taken at the Johns Hopkins Hospital in 1896 and 1897, when Dr. Pratt was a medical student. The long sixteen-page introduction presents an interesting combination of a biography and a character sketch. The illustrations preceding the text comprise a picture of the Class of 1898 with Drs. Osler and Hurd, three pictures of Dr. Osler, and a facsimile reproduction of a page of the original notes. Indexes of names and subjects conclude the book. It is recommended for all medical-history collections.

The Devil in Massachusetts: A modern inquiry into the Salem witch trials. By Marion L. Starkey, M.A. 8°, cloth, 310 pp. New York: Alfred A. Knopf, 1949. $3.50.

Miss Starkey has written an interesting narrative of the Salem witchcraft craze of 1692, and the resulting infamous witch trials. A selected bibliography and a good index conclude the volume. The book is well published and should be in all collections on witchcraft and allied delusions. The bobbysoxers of those days were largely responsible for this small epidemic of witch hysteria — small, because in the late middle ages in Europe and England, thousands of persons were put to death, in comparison with the twenty executions following the Salem trials.

NOTICES

ANNOUNCEMENT

Dr. Charles S. Gleason announces the opening of an office at 121 High Street, Wareham, for the practice of pediatrics.

BOSTON CITY HOSPITAL HOUSE OFFICERS' ASSOCIATION

A meeting of the Boston City Hospital House Officers' Association will be held in the Dowling Amphitheater of the Hospital on Tuesday, March 21, at 7 p.m. Dr. Arthur H. Blakemore will speak on the subject "Porta Caval Anastomosis." Drs. Claude E. Welch and Robert R. Linton will lead the discussion.

AMERICAN COLLEGE OF SURGEONS

A sectional meeting of the American College of Surgeons will be held in Montreal, Quebec, on March 20 and 21, with headquarters at the Mount Royal Hotel. Attendance will be largely from Maine, Massachusetts, New Brunswick, Newfoundland, New Hampshire, Nova Scotia, Ontario, Prince Edward Island, Rhode Island, Vermont and Quebec, although there is no geographic restriction. Two other sectional meetings will be held in Winnipeg, on April 3 and 4, and in Seattle on April 10 and 11.

The medical profession at large, medical students and hospital personnel, are invited to join with the fellows of the College in the meetings, which will be addressed by nationally prominent visiting and local speakers.

The sectional meeting will open at 8:30 both mornings with the showing of medical motion pictures. On the first morning these will be followed at 10:00 o'clock by a scientific session for the medical profession on "Arterial Lesions of the Extremities," "The General Adaptation Syndrome and the Diseases of Adaptation" and "Venous Thrombosis." At the same hour hospital personnel will hold a conference.

Luncheons will be held on both days, followed by round-table conferences on subjects presented at the preceding morning sessions. The panel discussions for medical personnel on the first afternoon will be on "Cesarean Section" and "Jaundice." Hospital personnel will meet concurrently. On the first evening a dinner for fellows, other members of the medical profession and hospital representatives will be held, after which there will be talks on activities of the College and a symposium on Cancer, at which "Melanoma," "Bronchiogenic Carcinoma" and "Brain Tumors — Differential Diagnosis" will be discussed.

On the second morning, after the showing of medical motion pictures, the medical program will be devoted to discussion of "Gastric and Intestinal Intubation," "The Management of Acute Head Injuries," "Internal Fixation of Fractures" and "Recent Advances in Anesthesia." Concurrent with the showing of medical motion pictures, papers on trauma will be presented on "Treatment of Shock in Trauma," "Treatment of the Commoner Hand Injuries" and "Present-Day Status of the Treatment of Burns." Panel discussions on "Surgery of the Hand" and "Surgical Lesions of the Stomach" will feature the afternoon medical program. The hospital personnel will hold conferences both morning and afternoon.

ROSWELL PARK AWARD

At an invitation dinner at the Hotel Statler, Buffalo, New York, on March 30, the Buffalo Surgical Society will award the Roswell Park Medal to Dr. Dallas B. Phemister for outstanding achievement. Dr. Phemister will deliver the Roswell Park Lecture, his subject being "Changes in the Treatment of Infections of Bones and Joints Since the Introduction of Antimicrobial Drugs."

SIXTIETH ANNIVERSARY OF CLARK UNIVERSITY

The sixtieth anniversary of the founding of Clark University will be commemorated on April 21. At 8:30 p.m. Miss Anna Freud will address herself primarily to psychiatrists and psychoanalysts on the subject "Early Feeding and Sleeping Problems in Infancy." The president and members of the Department of Psychology at Clark University invite any interested members to attend this lecture. In view of the fact that the demand for admission is expected to be great admission will have to be by ticket, and it is suggested that persons wishing to attend write prior to April 1 to Dr. Joseph Weinreb, Department of Psychology, Clark University, Worcester, Massachusetts, requesting tickets of admission.

SECOND EUROPEAN CONGRESS OF NATIONAL ASSOCIATIONS OF GASTROENTEROLOGY

The Second European Congress of National Associations of Gastroenterology will be held in Madrid, May 3 to 7, 1950. The topic of the Congress will be "Diseases of the Biliary Tract, except Lithiasis and Cancer." Distinguished leaders in the field of gastroenterology will discuss the various phases of the subject.

The sessions of the Congress will be held at the Consejo Superior de Investigaciones Cientificas, calle de Serrano, 119, where the General Secretary will be functioning from May 1 until the end of the Congress.

AMERICAN PHYSICIANS ART ASSOCIATION

The twelfth art exhibition of the American Physicians Art Association will be held in conjunction with the American Medical Association Convention at San Francisco Auditorium June 26 to 30, 1950.

Any physician who follows the hobby of fine or applied arts can exhibit at this convention by becoming a member of the American Physicians Art Association and applying for entry blanks and shipping labels of the secretary F. H. Redewill, M.D., 526 Flood Bldg., San Francisco 2, California.

AMERICAN CONGRESS OF PHYSICAL MEDICINE

The twenty-eighth annual scientific and clinical session of the American Congress of Physical Medicine will be held at the Hotel Statler, Boston, from August 28 to September 1. Scientific and clinical sessions will be given. All sessions will be open to members of the medical profession in good standing with the American Medical Association. In addition to the scientific sessions, the annual instruction seminars will be held on the first four days. These seminars will be offered in two groups. One set of ten lectures will consist of basic subjects and attendance will be limited to physicians. One set of ten lectures will be more general and will be open to physicians as well as to therapists registered with the American Registry of Physical Therapy Technicians or the American Occupational Therapy Association. Full information may be obtained from the American Congress of Physical Medicine, 30 North Michigan Avenue, Chicago 2, Illinois.

AMERICAN ASSOCIATION OF BLOOD BANKS

The third annual meeting of the American Association of Blood Banks will be held at The Stevens Hotel in Chicago on October 12, 13 and 14. The program is planned to attract blood-bank personnel, hospital executives, pathologists, clinicians, surgeons and others interested in the collection, preservation and administration of blood and blood derivatives.

Further information may be obtained from the Office of the Secretary, 3301 Junius Street, Dallas 1, Texas.

FILM ON CANCER DIAGNOSIS

Recently the American Cancer Society released the second film of a professional series on cancer diagnosis, entitled "Breast Cancer: The problem of early diagnosis." The film uses color, sound and animation to emphasize the crucial importance of early diagnosis. It is a 16-mm. film, and its running time is 34 minutes.

A print of this film is in the hands of the Massachusetts Division, Inc., of the American Cancer Society at 462 Boylston Street, Boston, and it is available for a single showing without cost to medical societies, medical schools, hospital staffs and any gathering of physicians or nurses.

SOCIETY MEETINGS AND CONFERENCES

OCTOBER 3–MAY 19. Massachusetts Department of Mental Health. Postgraduate Seminar in Neurology and Psychiatry. Page 286, issue of August 18.

JANUARY 9–DECEMBER 1. Laboratory Courses at Communicable Disease Center, Atlanta, Georgia. Page 950, issue of December 8.

MARCH 4–29. Springfield Hospital Educational Program. Page 347, issue of March 2.

MARCH 17 AND 18 (note change of dates). New England Health Education Association. Page 305, issue of February 23.

MARCH 20. Hartford Medical Society. Page 386, issue of March 9.

MARCH 20 AND 21. American College of Surgeons. Page 427.

MARCH 21. South End Medical Club. Page 385, issue of March 9.

MARCH 21. Boston City Hospital House Officers' Association. Page 427.

MARCH 22. Beth Israel Hospital Lecture. Page 270, issue of February 16.

MARCH 23. Cornell Alumni Day. Page 234, issue of February 9.

MARCH 24. Public Seminar in Chemotherapy. Page 270, issue of February 16.

MARCH 24. Society of American Bacteriologists, Northeast Branch. Page 385, issue of March 9.

MARCH 29. Beth Israel Hospital Lecture. Page 270, issue of February 16.

MARCH 30. Buffalo Surgical Society (see Roswell Park Award). Notice above.

APRIL 3. New England Cardiovascular Society. Page 724, issue of November 3.

APRIL 3. Hartford Medical Society. Page 386, issue of March 9.

APRIL 3 AND 4. American College of Surgeons. Page 427.

APRIL 7. Public Seminar in Chemotherapy. Page 270, issue of February 16.

APRIL 10 AND 11. American College of Surgeons. Page 427.

APRIL 13. The Value of the Bone Bank. Dr. Joseph H. Shortell. Pentucket Association of Physicians. 8:30 p.m. Haverhill.

APRIL 17. Boston Lying-in Hospital Obstetric Round Table. Page 888, issue of December 1.

APRIL 17. Hartford Medical Society. Page 386, issue of March 9.

APRIL 21. Sixtieth Anniversary of Clark University. Notice above.

APRIL 22–29. American Association of Industrial Physicians and Surgeons. Page 305, issue of February 23.

(Notices concluded on page xiii)

NOTICES (*Concluded from page 428*)

APRIL 24–28. National Tuberculosis Association. Page 678, issue of October 27.

APRIL 25–28. Philadelphia County Medical Society. Page 386, issue of March 9.

APRIL 26. New England Obstetrical and Gynecological Society. Rhode Island Hospital and Providence Lying-in Hospital, Providence, Rhode Island.

APRIL 29. Eastern Section of American Congress of Physical Medicine. Page 306, issue of February 23.

APRIL 29. Long Island College of Medicine Alumni Day. Page 348, issue of March 2.

MAY 3. Norfolk District Medical Society Anniversary Dinner.

MAY 3–7. Second European Congress of National Associations of Gastroenterology. Page 428.

MAY 4. Suffolk District Censors' Meeting. Page 234, issue of February 9.

MAY 14–19. International and Fourth American Congress of Obstetrics and Gynecology. Page 160, issue of January 26.

MAY 16–18. Massachusetts Medical Society. Annual Meeting. Hotel Statler, Boston.

MAY 17. Luncheon Meeting. Section on Diseases of the Eye, Ear, Nose and Throat, Massachusetts Medical Society. Hotel Statler, Boston. 12:00 m.

MAY 24–26. American Association of Genito-Urinary Surgeons. Page 234, issue of February 9.

JUNE 22–25. American College of Chest Physicians. Page 270, issue of February 16.

JUNE 24 AND 25. American Society for the Study of Sterility. Page 38, issue of January 5.

JUNE 26–30. American Physicians Art Association. Page 428.

JULY 17–22. International Congress for Scientific Research. Page xvii, issue of September 1.

AUGUST 21–26. International Society of Hematology. Page 888, issue of December 1.

AUGUST 28–SEPTEMBER 1. American Congress of Physical Medicine. Page 428.

AUGUST 31–SEPTEMBER 7. World Federation for Mental Health. Page 306, issue of February 23.

SEPTEMBER 3–9. International Heart Congress. Page 160, issue of January 26.

SEPTEMBER 27–29. Mississippi Valley Medical Society. Page 991, issue of December 15.

OCTOBER 12–14. American Association of Blood Banks. Page 428.

NOVEMBER 1. New England Obstetrical and Gynecological Society. Hotel Somerset, Boston.

DISTRICT MEDICAL SOCIETIES

NORFOLK

MARCH 28.
MAY 3. Anniversary Dinner.

SUFFOLK

MAY 4. Censors' Meeting.

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING THURSDAY, MARCH 23

THURSDAY, MARCH 23

*9:00–10:00 a.m. Grand Rounds. House of the Good Samaritan.

FRIDAY, MARCH 24

*9:00 a.m.–12:00 m. Combined Medical and Surgical Staff Rounds. Peter Bent Brigham Hospital.

*1:30 p.m. Tumor Clinic. Out-Patient Department, Mount Auburn Hospital, Cambridge.

MONDAY, MARCH 27

*11:30 a.m.–12:15 p.m. Chest X-Ray Conference. South End Health Unit, 57 East Concord Street, Boston. Dr. Cleaveland Floyd in charge.

*12:15–1:15 p.m. Clinicopathological Conference. Main Amphitheater, Peter Bent Brigham Hospital.

5:15 p.m. Staff Meeting. Harris Hall, New England Deaconess Hospital.

TUESDAY, MARCH 28

*12:15–1:15 p.m. Clinicoroentgenological Conference. Peter Bent Brigham Hospital.

*1:30–2:30 p.m. Pediatric Rounds. Burnham Memorial Hospital for Children, Massachusetts General Hospital.

WEDNESDAY, MARCH 29

*11:00 a.m.–12:00 m. Medical Rounds. Children's Medical Center.

*12:00 m. Lecture. Speaker to be announced. Margaret Jewett Hall, Mount Auburn Hospital, Cambridge.

*12:00 m.–1:00 p.m. Clinicopathological Conference (Children's Medical Center). Amphitheater, Peter Bent Brigham Hospital.

*2:00–3:00 p.m. Combined Clinic of all Medical Center Services. Amphitheater, Children's Hospital.

*7:30 p.m. Modern Management of Syphilis. Dr. G. Marshall Crawford. Beth Israel Hospital.

*Open to the medical profession.

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